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VOL. II.—PART I.

MANUAL

OF

PATHOLOGICAL HISTOLOGY

BY

CORNIL AND RANVIER

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By A. M. HART

Vol. II.

SPECIAL PATHOLOGICAL HISTOLOGY

LESIONS OF THE ORGANS

PART I.

WITH 125 FIGURES INTERSPERSED IN THE TEXT

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CONTENTS.

PART III.

DISEASES OF THE ORGANS.

Section I.—The Respiratory System.

CHAPTER I.

CHAPTER II.

GENERAL PATHOLOGICAL HISTOLOGY OF THE RESPIRATORY SYSTEM.

CHAPTER III.

SPECIAL PATHOLOGICAL HISTOLOGY OF THE RESPIRATORY SYSTEM.

V. LESIONS OF THE LUNG—*continued.*

SECTION II.—THE DIGESTIVE SYSTEM.

CHAPTER I.

THE MOUTH.

CHAPTER II.

THE TONSILS.

CHAPTER III.

THE SALIVARY GLANDS.

CHAPTER IV.

THE PHARYNX AND ŒSOPHAGUS.

CHAPTER V.

THE STOMACH.

CHAPTER VI.

THE INTESTINE.

MANUAL

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PART III.

DISEASES OF THE ORGANS.

Section I.—The Respiratory System.

CHAPTER I.

NORMAL HISTOLOGY OF THE RESPIRATORY SYSTEM.

The different parts of the respiratory system are named **the larynx, the trachea, the bronchi,** and **the lungs.** The latter are enveloped by the pleuræ. In entering the larynx air passes by way of the nasal fossæ, or the mouth, through the pharynx. The mouth and the pharynx belong to the alimentary canal, the lesions of which we shall study later. We shall treat here the nasal fossæ, as we consider them to belong to the respiratory apparatus. We will first briefly recall in a few words the normal histology of these different parts.

The nasal fossæ consist of different parts, the structure of which varies; first, the vestibule of the nasal fossæ, which is furnished with rather coarse hairs and lined with a stratified pavement epithelium; secondly, the nasal fossæ properly so called, which may be divided into a respiratory and an olfactory region, which latter corresponds to the upper part of the septum, the superior turbinated bone, and part of the middle turbinated bone; thirdly, and finally, the sinuses.

21

The whole mucous membrane of the respiratory tract—that of the sinuses as well as of the nasal fossæ—is lined by a ciliated cylindrical epithelium. It is very thick and highly vascular, except in the sinuses, where it is thin and intimately attached to the bony surfaces, which it covers. It contains numerous mucous glands. The olfactory region is particularly remarkable for the terminations of the olfactory nerves, which are peculiar epithelial cells, elongated, nucleated, and terminating in a rod-like extremity (*Schultze*). These olfactory cells are situated between ordinary cylindrical cells, and in this region the mucous membrane is thicker than in others. It is yellowish in colour, and contains simple or ramified utricular glands, generally elongated in shape, the cells of which are infiltrated with yellow or brown pigment granules.

The larynx, trachea, and **bronchi** present for study a mucous membrane which lines their internal surfaces, a fibro-cartilaginous framework, smooth and striated muscles, and vessels and nerves.

The mucous membrane is lined by an epithelium, which is tesselated and stratified on the epiglottis and on the inferior vocal cords, and cylindrical and ciliated in all the rest of the laryngeal cavity, in the trachea, as well as in the bronchi; and, finally, a squamous epithelium lines the pulmonary acini. The corium is composed of two layers. The first, situated immediately under the epithelium, is composed, in great measure, in the larynx and trachea of elastic fibres limited by a homogeneous layer 11 μ thick, called the basement membrane, in which are implanted the cylindrical cells of the epithelial lining. On the most prominent part of the inferior vocal cord the mucous membrane shows numerous papillæ. These papillæ, well described by Coÿne, are similar to those on the palmar suface of the fingers; the epithelium covering them is tesselated, and beneath the epithelium is a limiting membrane. Elsewhere the superficial part of the corium of the larynx is formed of retiform tissue (Coÿne); this latter contains a certain number of lymphatic follicles, situated in man in that part of the mucous membrane which lines the ventricles of the larynx: their number is from thirty to fifty, and their form is variable, often ovoid. The second layer of the mucous corium is composed of connective and elastic tissue which unites it to the muscular and cartilaginous layers, and in it are lodged the acinous glands. The numerous **mucous or acinous glands** of the larynx, trachea, and bronchi are racemose glands.

Their openings are easily distinguished even with the naked eye. A large number of them are arranged in the form of the letter L, the horizontal arm of which envelopes the cartilage of Wrisberg, and the longitudinal arm is pushed into the larynx in front of the arytenoid cartilages. The round saccules of these glands contain mucous cells, with a flat nucleus at their base. Their ducts are lined by cylindrical epithelium.

The fibro-cartilaginous framework of the larynx consists of the thyroid, cricoid, and arytenoid cartilages, formed of ordinary cartilaginous tissue, and of the epiglottis and the cartilages of Santorini and Wrisberg, composed of reticulated cartilage. The elastic fibres of the matrix of these various cartilages are continuous with those of the mucous membrane. The rings of the trachea, as well as the angular and irregular plates of the bronchi, are composed of ordinary cartilaginous tissue. All the cartilages of the larynx in children are also composed of the same tissue. The ligaments which unite the various parts are chiefly composed of elastic tissue. The middle crico-thyroid and the inferior thyro-arytenoid ligaments owe their yellow colour to the great abundance of this tissue.

The muscles which move the various parts of the respiratory apparatus are striated in the larynx and smooth in the trachea and the bronchi. They complete the circle of the trachea, and posteriorly are inserted into the ends of the cartilaginous rings. In the bronchi they form complete circles, which are still visible in bronchi 0·2 mm. in size. Moleschott even asserts the existence of smooth muscle-fibres in the walls of the pulmonary vesicles, a fact which is denied by Kölliker and most anatomists. The distribution of the blood-vessels in the various layers of the respiratory mucous membrane is in no way peculiar; they form a superficial capillary network. The inferior laryngeal nerve is chiefly composed of large nerve-tubes, and the superior laryngeal nerve of small nerve-tubes. The latter terminates in the mucous membrane, and forms a deep and a superficial network of pale fibres, among which small microscopic ganglia are found.

The bronchi may be divided as follows:—1. Large bronchi continuous with the trachea. 2. Bronchioles, which are more than 1 mm. in diameter. 3. Sublobular and intralobular bronchi, which penetrate into the lobules and break up into terminal alveoli. The large and medium-sized bronchi have four coats, an external fibrous coat, which contains the bronchial cartilages and the glands, a muscular coat, an internal fibrous coat;

and, finally, the mucous coat lined by cylindrical ciliated epithelium. In the intralobular bronchi the external coat is thinner, and contains neither cartilaginous plates nor glands. It is in immediate relation with the fibrous tissue of the neighbouring alveoli; the muscular coat is relatively rather thick; the internal fibrous coat is rich in elastic fibres, and is continuous with the mucous coat. This latter, in a condition of semi-dilatation or contraction of the bronchial tubes, falls into longitudinal folds, so that in transverse sections the lumen of the tubes shows a festooned edge. In all these divisions of the bronchi the mucous corium is bounded, as in the trachea and larynx, by a rather thick, transparent and homogeneous basement membrane, in which are implanted the cylindrical cells. The intralobular bronchi divide dichotomously to form the terminal bronchi. These contain only a few muscle-fibres scattered in a single coat which is rich in elastic fibres. The epithelium here loses its cilia, and becomes cubical and more and more flattened. When it reaches the lobule the ultimate bronchus becomes suddenly contracted, then swells out in the shape of a funnel, which is called the infundibulum. From the latter spring three to five divisions, which are the alveolar ducts into which the pulmonary alveoli open. The whole, composed of the ultimate bronchus, the infundibulum, the air-tubes, and the alveoli, is called a **pulmonary lobule.**

The lungs are composed of lobes and of lobules. The latter are nothing else than a group of pulmonary infundibula in which the intralobular bronchi terminate; hence the most important part of the lung structures are the ultimate bronchial ramifications and the infundibula. An infundibulum is composed of a group of alveoli opening into a common cavity, in which an ultimate bronchus terminates. The alveoli, the mean diameter of which is 0·20 mm., are round or polygonal. They may be found not only grouped round an infundibulum, but isolated, and suspended to the short terminal bronchus. In a delicate section made from an inflated and dried lung, the alveoli appear in the form of round cavities, limited by septa composed of connective tissue and elastic fibres. This fibrous framework of the lung is very extensible; it is continuous with the wall of the ultimate bronchi, and serves to support the vascular network and to give insertion to the epithelium which lines the alveoli.

The pulmonary epithelium is composed in the terminal bronchi of small tesselated cells, very regular in size and easily seen; and on the surface of the alveoli of squamous cells, the

presence of which can be easily demonstrated in preparations stained with nitrate of silver. By means of this reagent (figs. 1 and 2) the epithelial cells of the lung in the frog, in mammalia, and in new-born infants can be plainly demonstrated. These cells line the whole extent of the alveoli, but their nuclei are always situated within the meshes of the capillary network.

The blood-vessels of the lung are derived from two sources, the bronchial arteries, whose rôle is principally nutritive, and the pulmonary artery, which is more particularly destined to carry on hematosis. The ramifications of the pulmonary arteries accompany the divisions of the bronchi and penetrate with them into the lobules. There they divide into capillaries and form in the

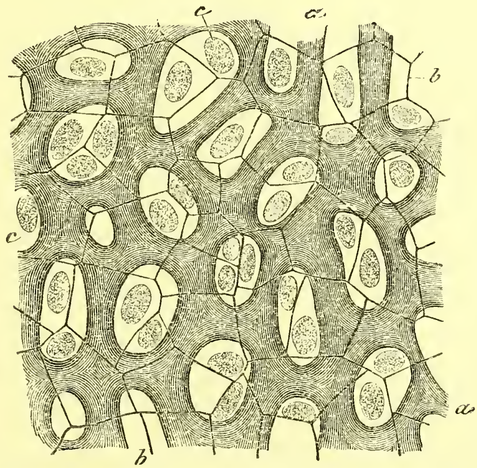


FIG. 1.—SEPTA AND EPITHELIUM OF THE LUNG OF THE FROG, AFTER KÖLLIKER.

a, injected pulmonary vessels ; *b*, borders of the epithelial cells stained black with nitrate of silver ; *c*, nuclei of the cells. Magnified 200 diameters.

interalveolar septa a network of very fine meshes, 4 to 18 μ in diameter, the vessels of which measure from 6 to 11 μ in width. When the alveoli contract or partly dilate, these vessels become tortuous, and project into the alveolar cavity, raising the epithelium.

The lymph-vessels of the lung are very numerous ; the deeper spring from the bronchial mucous membrane, the adventitious coat of the blood-vessels, particularly of the pulmonary arteries, and also, according to Wiwodzoff, from the walls of the pulmonary vesicles. They surround the bronchi and the pulmonary arteries, forming more or less complete sheaths. On the surface of the

normal lung the pulmonary veins and the lymph-vessels may be seen in the form of polyhedral figures which correspond to the base of the lobules. These vessels are surrounded by a certain amount of connective tissue. In sections of the lung the lobules, limited by their envelope of connective tissue, may be recognised under a low power. In these perilobular sheaths the pulmonary veins and the lymphatics ramify. In the centre of the lobule may be found, on the other hand, the intralobular bronchus and its ramifications; the lobular division of the pulmonary artery is attached to it as well as the lymph-vessels. These vessels are surrounded by a certain amount of connective tissue, which forms a sheath and accompanies them in their divisions and subdivi-

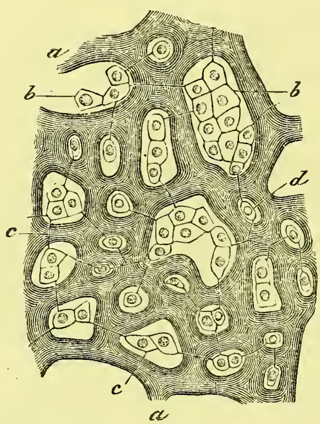


FIG. 2.—SEPTA AND EPITHELIUM OF THE LUNG OF A SMALL MAMMAL, AFTER KÖLLIKER.

sions into the interior of the lobule. It is important to understand this arrangement, so as to localise the lesions of bronchopneumonia and to understand the position of pulmonary tubercles, as we shall see later on.

The pleura, the serous membrane of the thoracic cavity, is composed of two parts. The first or visceral covers the surface of the lung; it is thin and composed of a layer of loose connective tissue, and is lined on its external surface by flat epithelium cells; the other or parietal has on its free surface similar cells, and on its deep surface it adheres to the ribs and intercostal muscles. The parietal pleura is thick, and is composed of two layers of connective tissue. The first, composed of loose connective tissue, is situated under the epithelium; the other, distinctly fibrous, contains

a large quantity of elastic fibres. On the edges of the lung the visceral pleura has villous processes. This membrane contains blood-vessels. Its lymph-vessels, which are abundant, have been studied by Dibkowski; they communicate directly with the serous cavity by open pores between the endothelial cells; these pores or stomata are particularly met with in the parietal pleura between the ribs.

To recapitulate, the essential part of the lung, where the exchange of gas between the air and the blood takes place, is the pulmonary alveolus. From the most accepted morphological point of view the pulmonary alveolus, lined with flat cells and formed of a connective tissue and elastic membrane, corresponds to a mesh of connective tissue or to a small serous cavity, which may be considered as identical. Though the alveolar cavity is in contact with the atmospheric air, the cellular elements which constitute its walls do not become decomposed, for it is never completely empty and always contains air saturated with vapour; hence the epithelial cells of the lung never become dry. The pathological anatomy of the lung is reduced essentially to that of the pulmonary alveolus.

CHAPTER II.

GENERAL PATHOLOGICAL HISTOLOGY OF THE RESPIRATORY SYSTEM.

Before proceeding to give a description of each of the diseases of the respiratory system, the histological changes in their essential parts should first be studied from a broad point of view. From this point of view the respiratory system may be divided into two distinct portions, the air-tubes and the lung. The diseases of the air-tubes, from the nasal fossæ to the termination of the bronchi in the pulmonary alveoli, are for the most part independent of diseases of the lung. The latter, however, always attack the final terminations of the bronchi in the same degree and in the same way as the pulmonary alveoli; the ultimate bronchial ramifications belonging in reality to the lung by their structure and their pathology. There are, moreover, diseases which, like tubular pneumonia or broncho-pneumonia, affect at the same time the lung and the bronchi of all sizes, even their final divisions. Finally, infectious diseases and generalised neoplasms invade in different degrees all segments of the respiratory tract. With these exceptions the lesions of the air-tubes (nasal fossæ, larnyx, trachea, and bronchi) may be considered apart from those of the lung.

I. General Pathological Histology of the Air-tubes.

We will now pass in review the changes of the most essential parts composing these tubes—the epithelial lining, the glands, the mucous corium, and the smooth muscles.

A. Histological changes in the epithelial lining of the air-tubes. —The epithelial cells which line the mucous corium of the air-tubes are almost always cylindrical throughout. In the greater part of these passages—in the larnyx, the trachea, and the large bronchi, for example—it may be observed in mammals and in man that, above the basement membrane of the corium, there is nor-

mally a layer of small round cells and a superficial layer of cylindrical cells furnished with a plate surmounted by cilia; between these latter cells caliciform cells are disseminated. Fig. 3 shows the arrangement of the epithelial lining of the normal mucous membrane of the trachea in the rabbit.

Experimental inflammation.—Changes which take place in the epithelial lining of the air-tubes may be easily studied in inflammation experimentally induced in animals. To this end cantharidin[1] is injected under the skin. This irritating substance has the advantage of inflaming the tissues by means of the blood, hence its action is exercised on the deep layers in contact with the capillaries, and extends to the superficial layers. The conditions of spontaneous inflammation are by this means better realised than when irritating agents are applied to the surface of the mucous membrane. Two hours after the injection of a sufficient quantity of cantharidin into the subcutaneous cellular tissue of the rabbit, the mucous membrane of the trachea becomes uniformly red, but shows no abnormal secretion of mucus. In fine sections of the preparation hardened by the successive action of Müller's fluid,

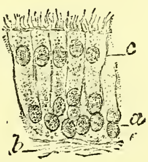


FIG. 3.—SECTION OF THE EPITHELIAL LAYER OF THE TRACHEA OF THE RABBIT IN THE NORMAL CONDITION.

b, superficial part of the mucous corium; *a*, layer of small cells in contact with the mucous corium; *c*, cylindrical ciliated cells. Magnified 250 diameters.

gum, and alcohol, the first phases of inflammation may be observed. The epithelial lining of the tracheal mucous membrane will be seen to be thicker than normally, and almost everywhere many layers of small round cells may be seen at its base, instead of the single layer which exists there normally. The superficial cells still retain the cylindrical form. In the parts where the inflammation is more intense all the epithelial lining is composed of cells, round or polyhedral by reciprocal pressure, about the size of lymph cells, and the cylindrical cells of the surface have disappeared. The superficial round cells still almost always retain

[1] In employing subcutaneous injections of cantharidin we use a solution of this substance in acetic ether. With 4 to 6 grammes of the solution at the degree of concentration which is obtained at the temperature of 20° centigrade—that is to say, with a dose of from 5 to 8 mgrs. of cantharidin—symptoms of intense poisoning are induced in the rabbit, and death occurs in a few hours.

their cilia (see *c*, fig. 4); these, however, are not arranged regularly on the plate of a cylindrical cell, but are inserted without order. When the inflammation is intense the epithelial lining is much thickened, and appears in sections in the form of tumefied projections. At the base of these projections above the basement membrane are seen many close layers of round cells, then vacuoles filled with a fluid containing granules and lymph cells. Fusiform flattened cells with ovoid nuclei, everywhere mixed with lymph cells, limit these vacuoles. The epithelial surface is composed of cylindrical or round ciliated cells (see fig. 5). It is evident that the round or lymph cells, derived in a great measure from the capillaries of the mucous and submucous tissue, have displaced forwards and separated the pre-existing and fusiform cylindrical cells. The ciliated cylindrical cells have themselves become changed in shape; after division of their nucleus and protoplasm

FIG. 4.—SECTION OF THE MUCOUS MEMBRANE OF THE TRACHEA IN A CASE OF INFLAMMATION INDUCED BY CANTHARIDIN. THE SECTION WAS MADE AFTER HARDENING THE PREPARATION IN OSMIC ACID.

b, surface of the mucous corium; *a*, round cells situated at the base of the epithelial lining; *a'*, the same cells occupying almost the whole extent of the section of the epithelial lining; *c*, layer of round cells furnished with cilia. Magnified 250 diameters.

they are transformed into round cells, the most superficial of which still preserve their cilia.

When the artificially induced inflammation is more intense, instead of simply causing an accumulation of cells on the surface of the mucous membrane, the epithelium is rapidly desquamated. Its place is taken by a layer of lymph cells, which, mixed with the fluid exuded from the vessels, form a muco-purulent secretion on the surface of the mucous membrane. If, for example, 50 centigrammes of a 1 per cent. solution of nitrate of silver be injected into the trachea of a rabbit, and if two hours afterwards the trachea be placed in a hardening fluid, so that sections can be cut, a layer of lymph cells, more or less pressed together, and taking

consequently the shape of small cubic blocks, will be found in the place of the normal epithelial lining. In this experiment the irritating nitrate of silver first attacks the superficial cells, and it is natural that they should be desquamated more quickly and thoroughly than in the previous experiment. The irritation caused by the nitrate of silver being, moreover, more intense, the superficial layer of the mucous corium is infiltrated by a great number of lymph cells. If the artificially induced inflammation caused by cantharidin or by nitrate of silver be exerted, not on the trachea or on the larger bronchi, but on the small bronchi, the

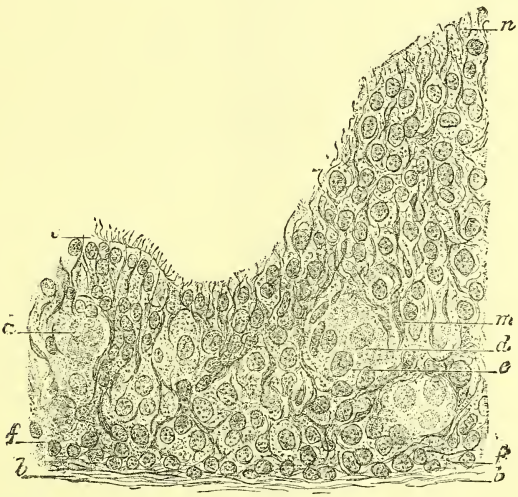


Fig. 5.—Section of Epithelial Lining of the Trachea of the Rabbit, from a case of poisoning by Cantharidin, obtained after hardening the preparation in Osmic Acid.

b, superficial part of the mucous corium ; *f*, small cells in contact with the latter ; *d*, *e*, lymph-cells contained in the vacuoles, round which may be seen flattened fusiform cells, or cylindrical cells, *m* ; *c*, cylindrical superficial cells furnished with cilia. Magnified 250 diameters.

newly formed cells, instead of simply causing a thickening of the epithelial lining, as in the large tubes, completely fill the lumen of the small bronchi.

Catarrhal inflammation of the air-tubes.—The preceding experiments have enabled us to understand what takes place in the epithelial lining in catarrhal or superficial inflammation in man. The phenomena observed vary according to the intensity of the inflammation. In slight inflammation, when only an increase of the mucous secretion and congestion of the corium are observed by the naked eye, the epithelial lining remains intact, and no

new cells are formed; but the superficial cells contain and secrete a larger quantity of mucus than normally. A large quantity of the superficial cylindrical cells of the mucous membrane are caliciform. They may be observed isolated in the mucus which covers the free surface of the mucous membrane, and also in sections of this membrane. They are oval or globular in form, transparent, mucous, limited by a delicate membrane which is often wrinkled or plaited. The free end of these cells has neither plate nor cilia. The nucleus, more or less deformed, sometimes crescentic in shape, is found pushed towards the lower extremity of the cell. In sections of slightly inflamed mucous membrane, whether it be of the trachea (as in fig. 7) or of a medium-sized bronchus (as in fig. 8), ciliated and caliciform cells are seen to be arranged in an alternate manner. The latter are as numerous, often even more numerous, than the ciliated cells. They themselves have no cilia, and are surrounded by ciliated cells thinned by

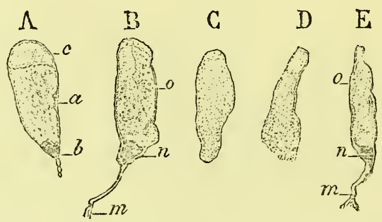


FIG. 6.—MUCOUS OR CALICIFORM CELLS FROM THE SURFACE OF AN INFLAMED HUMAN TRACHEA.

A, mucous cell, of which the nucleus, *b*, is small, and situated near the tail of the cell. The protoplasm is mucous, and the free border of the cell is surmounted by a drop of mucus, *c*. B, C, D, E, isolated mucous cells of various shapes; *m*, *m*, filaments of insertion of the tail of these cells; *n*, *n*, nuclei; *o*, mucous protoplasm. Magnified 300 diameters.

pressure. The caliciform cells participate in the catarrhal secretion of the air-tubes in subacute inflammation. This lesion resembles that observed in the ducts and saccules of the acinous glands of the mucous membrane of the air-passages under the same pathological conditions. It is simply an exaggeration of the physiological phenomenon. This mucous condition of a large number of epithelial cells is not necessarily accompanied with the new formation of cells, nor with diapedesis of white blood corpuscles into the deep layers of the epithelial investment. The round cells situated upon the basement membrane are not always more numerous than in the normal condition.

Though in very slight catarrhal inflammation, which is only revealed by an increased secretion of mucus, the deep layer of the

epithelium and the corium remains intact, it is no longer the case if the inflammation becomes more acute. When in-

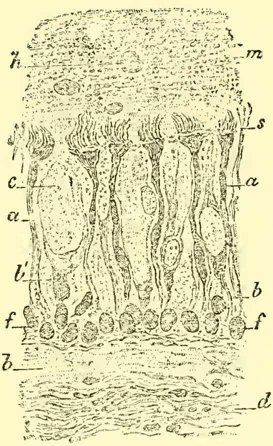


FIG. 7.—SECTION OF THE EPITHELIAL LINING OF A TRACHEA AFFECTED WITH CATARRHAL INFLAMMATION.

d, corium; *b*, basement membrane; *f*, *f*, round cells in connection with the basement membrane; *c*, caliciform cells, of which the nucleus, *b'*, has been pushed to the lowest part, near to their base of implantation, *b*; narrow, compressed, cylindrical cells with flattened nuclei, *a*, and cilia, *s*; *m*, free mucus on the surface; *h*, balls of mucus. Magnified 300 diameters.

flammation of the mucous membrane of the air-passages has existed some days, and has reached its climax, as may be observed

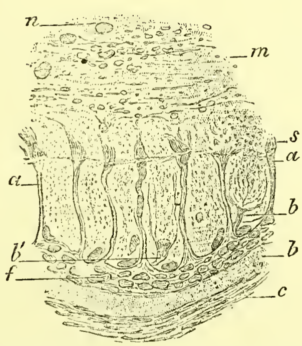


FIG. 8.—SECTION OF THE EPITHELIAL LINING OF A BRONCHUS AFFECTED WITH CATARRHAL INFLAMMATION.

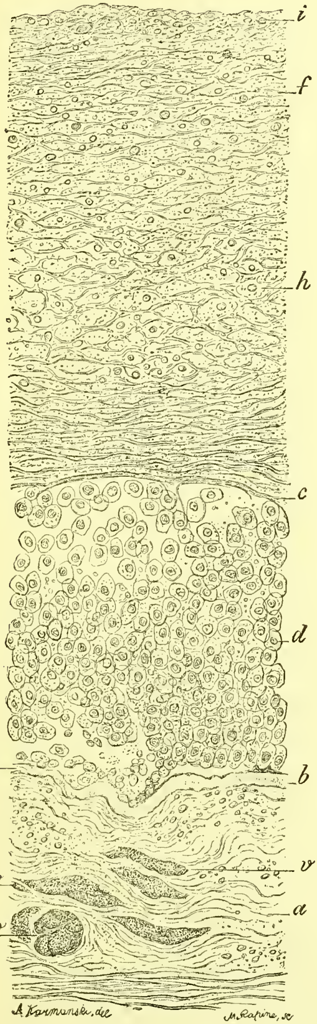
c, corium; *b*, basement membrane; *f*, round cells contiguous to the basement membrane; *b*, *b'*, caliciform cells; *a*, flattened cylindrical cells with cilia, *s*; *m*, mucus; *n*, mucus-balls. Magnified 300 diameters.

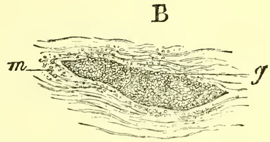
in subjects who succumb either to broncho-pneumonia, typhoid fever, cardiac disease, or phthisis, which is almost always accompanied with laryngo-tracheal bronchitis, the epithelial lining and

the superficial layer of the mucous corium are found altered to a variable degree. The ciliated cylindrical cells are sometimes preserved, sometimes replaced by long, narrow, squamous cells implanted more or less regularly on the surface of the mucous membrane. Beneath this superficial layer are found either ovoid cells, round cells, or lymph cells deposited in one or more layers. Frequently all the cells of the epithelial lining fall into the secreted fluid, and only a layer of lymph cells remains in contact with the basement membrane; the latter, which is rather thick and homogeneous, is preserved, and presents its normal appearance. As long as it persists the surface of the mucous membrane does not appear ulcerated, notwithstanding all the lesions of the superficial epithelium. It may seem rough and irregular, but there is no loss of substance appreciable to the naked eye. Between the connective tissue and elastic fibres which are situated under the basement membrane there are always found a rather considerable number of lymph cells, while normally only flat cells are found here. In all catarrhal inflammation of a certain intensity this inflammatory lesion of the mucous corium is constant; it primarily affects only the superficial layer. The mucous membrane then appears to be slightly thickened, but this thickening is so slight that it more often passes unobserved, for the lesions of catarrhal inflammation of the air-passages can only be detected under the microscope; by it alone can multiple and rather deep lesions be appreciated, while with the unaided eye, trusting only to the more or less red discoloration of the mucous membrane, it is impossible to determine, at least in certain cases, if the appearances are produced by post-mortem blood stains or by actual inflammation.

In very acute inflammation of the mucous membrane, cells, which are sometimes colossal in size, develop in the epithelium. These changes in the epithelium are seen in their highest degree in inflammation which accompanies the eruption of variolous pustules in the larynx, trachea, and bronchi. In sections of pustules of the trachea, the swelling of which may be appreciated during the first three or four days of the eruption, the following is what may be observed from the basement membrane to the corium: first, a thick layer formed of round or slightly compressed lymph cells deposited one above another, or of rather large cells, of various shapes, round and polygonal, containing a rather large ovoid nucleus in the midst of a granular protoplasm (A, *d*, fig. 9); sometimes red blood corpuscles are present between them; secondly, a thick layer (A, *h*, fig. 9) formed of very fine

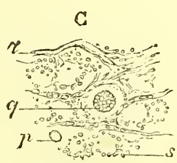
filaments of fibrin, woven into a close network, in the meshes of which are found granular lymph cells, red blood corpuscles, and masses of micrococci granules; this is, in fact, an actual fibrinous





B. Section of a Vessel of the Corium containing a Thrombus, which is composed of Lymph Cells and Granules of Micrococci.

Magnified 350 diameters.



C. Section of a Diphtheritic False Membrane.

r, fine fibrils of fibrin; *g*, lymph cells; *p*, red blood corpuscle. Magnified 350 diameters.

Fig. 9.—A. Section of an Inflamed Trachea in Variola.

a, connective tissue of the mucous membrane, in which the vessels, *v*, *v*, are obliterated by lymph cells filled with micrococci; *b*, basement membrane; *d*, lymph cells and indifferent cells forming a thick layer on the surface of the corium; from *c* to *i* is seen a fibrinous false membrane, in which the fibrils, *f*, limit meshes in which may be found red blood corpuscles and lymph cells, *h*. Magnified 200 diameters.

pseudo-membrane. At the base of the pustules the basement membrane is thinned, irregular, and even interrupted at places;

the submucous fibro-elastic tissue is filled with lymph cells, and it is hence comprehensible how easily these elements pass from the submucous connective tissue into the pustule. On the internal surface of the capillaries and the small veins (A, *v*, fig. 9, and B, *g*, fig. 9) may sometimes be seen an adhesive layer of lymph cells and groups of micrococcus, and the vessels may even become

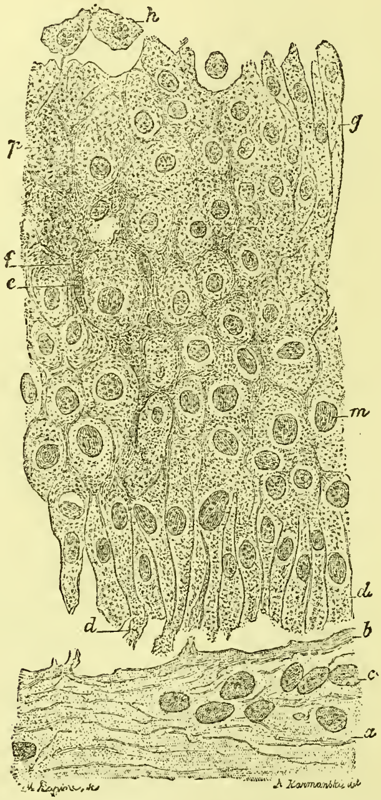


FIG. 10.—SECTION OF THE MUCOUS MEMBRANE OF THE TRACHEA IN VARIOLA.

a, tissue of the corium infiltrated with lymph cells, *c*; *b*, basement membrane; *d*, *d*, cylindrical cells implanted in the latter; *m*, lymph cells seated in the midst of granules in small lacunar spaces, which are bordered by flattened atrophied cells, containing one oval nucleus, *e*; *p*, granules; *h*, free cells on the surface of the epithelium. Magnified 350 diameters.

choked by these elements. Around the pustules the mucous membrane simply shows inflammatory lesions; lymph cells are present in great numbers in the deep layer of the epithelium and between the cylindrical cells; the epithelial cells, separated more or less in places by groups of lymph cells (see fig. 10), constitute

a membrane of considerable thickness; they are also irregular in shape and arranged without order on the free surface of the mucous membrane. When the variolous eruption is confluent in the air-passages, the fibrinous false membrane which caps the pustules uniformly covers more or less considerable segments of the internal surface of the tubes; it may extend to the larynx, the whole trachea, or to the large bronchi.

To recapitulate, the epithelial investment of the mucous membrane of the air-passages undergoes various changes in the acute stage of catarrhal inflammation; at the beginning, the pre-existing cylindrical cells are separated from one another and are compressed by lymph cells interposed between them, which also displace them forwards. If the inflammation is intense the epithelium is desquamated, and fibrin, exuded from the vessels with the lymph cells, sometimes becomes the starting-point of a false membrane, as may be seen in diphtheria, variola, and typhoid fever. When the inflammation is less acute, thick layers of elongated cells, which show a tendency to take the cylindrical form, may be seen in the epithelial lining. On recovery the layer of cylindrical cells is reconstituted as in the normal condition.

In subacute or chronic inflammation, such as occurs in cardiac affections or in tuberculosis, the epithelial lining undergoes various changes. Sometimes it is composed of a mass of irregularly cylindrical or oval cells. These cells do not form a regular level layer on the surface, as in the normal condition, but they are irregularly implanted by one of their extremities while they are free at the other. The surface of the mucous membrane, instead of being bordered by a regular line, formed by the plates of the cells with their cilia, is irregular and bordered by fusiform or cylindrical cells. In other cases the epithelium is almost entirely desquamated, and above the basement membrane there remains simply a layer of small oval cells implanted perpendicularly on the membrane parallel to one another. When tubercular granulations have existed only a short time in the submucous tissue, the epithelial investment at the level of the tubercles is almost intact, or else affected by inflammatory lesions. It is always present, though perhaps more or less altered, so long as the basement membrane is not destroyed and the tubercle not in the condition of ulceration. In cases, however, of old fibrous tubercle the bronchi and bronchioles, which traverse tissues which are themselves greatly altered, are lined with a

perfectly normal epithelium. In dilated bronchi the cavity is lined either by normal or by inflamed epithelium.

B. **Histological changes in the mucous glands of the air-passages.**—In inflammation, the glands of the air-passages always secrete a much larger amount of mucus than normally. On squeezing the mucous membrane this mucus, which is more or less opaque, may be seen to issue from the orifices of the glands. If, after opening the trachea and the bronchi, the cartilaginous rings be straightened, so that the mucous membrane is compressed against the fibro-cartilaginous plane, small drops of mucus or muco-pus may be squeezed from the excretory ducts of the glands. On removing these drops on the point of a needle and examining them under the microscope, they will be seen to be composed of normal cylindrical cells derived from the excretory duct of the gland, of cells full of mucus, of balls of free

Fig. 11.—Epithelial Cells lining a Dilated Bronchus affected with Chronic Inflammation.

b, lymph cells lodged between the fibres of the corium; *m*, basement membrane; *a*, fusiform cell, resembling somewhat a cylindrical cell; *d*, cell thinned towards its free end, implanted on the basement membrane by a pointed extremity. Magnified 250 diameters.

mucus, and of a more or less considerable quantity of lymph cells; these elements are suspended in a fluid which contains refractile granules and filaments of mucin. On wiping away the small drops of mucus exuding from the orifices of the glands these openings appear so patent and large that they might be mistaken for superficial erosions; but there is nothing of the kind, for in sections made perpendicular to the surface of the mucous membrane and the rim of the gland, the basement membrane is seen to be intact beneath the epithelial investment. The following details may be observed in sections: the mucus which fills the excretory ducts of the glands brims over on to the mucous membrane in the form of a mushroom; this plug of mucus contains cylindrical cells, lymph cells, and refractile balls, the nature of which is still disputed. The wall of the duct is lined with cylindrical cells, which are generally goblet-shaped and filled with mucus. In the subdivisions of the duct may

be found similar mucous contents and a similar epithelial lining. In the saccules the mucous cells undergo no change if the inflammation is only slight (see fig. 12, A); but in the lumen of the acinus or between the peripheral cells a few lymph cells are found. The glandular lesions are much more marked when the inflammation is more acute. The drops of mucus exuded from their ducts have a purulent appearance and contain, besides desquamated cylindrical cells, a large number of lymph cells; in microscopic sections the latter are seen in the ducts and in the lumen of the saccules as well as between the

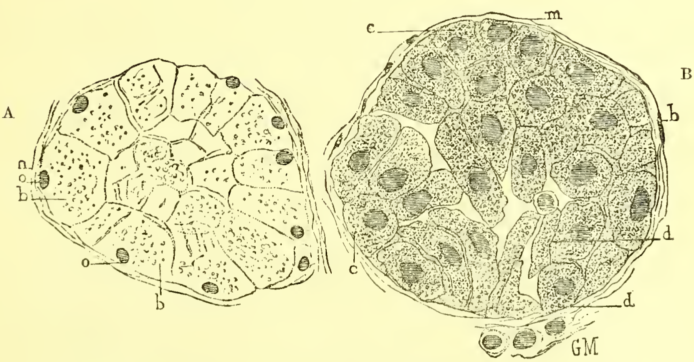


FIG. 12.—A. NORMAL SACCULE OF AN ACINOUS GLAND.

b, *b*, cells of clear mucus which contain refractile granules; *o*, nucleus of these cells. It is oval, small, and situated near the wall of the saccule. Magnified 300 diameters.

B. GLANDULAR SACCULE, THE CELLS OF WHICH HAVE BEEN GREATLY CHANGED BY INFLAMMATION.

c, *c*, prismatic or polyhedral cells implanted on the hyalin wall, *m*, of the saccule; *d*, cells flattened by pressure. These cells completely fill the saccule; they are large and irregular, and contain an opaque granular protoplasm and a large oval nucleus. Magnified 300 diameters.

mucous cells. The mucous cells of the acini themselves have also often undergone a peculiar change; their mucous contents are replaced by a granular protoplasm; the nucleus, which is originally small, becomes much larger, and instead of being situated at the periphery of the cell is found near the centre (see fig. 12, B). This modification of the cells, which is often limited to only some of the saccules of a gland, is always observed in very acute inflammation and in all chronic inflammation of the acinous glands of the air-passages.

While the ducts and saccules of the glands show the appearances just described, the surrounding connective tissue becomes

infiltrated with lymph cells, the number of which is in relation to the violence of the inflammation.

Inflammation of the air-passages, which occurs during the eruptive stage of variola, in idiopathic diphtheria of the larynx or in laryngo-typhus, is always of an acute type; in these diseases, moreover, the changes which take place in the mucous cells of the saccules, and the abundance of lymph cells exuded into the connective tissue and the glandular ducts, can be observed with great facility. The structure of the mucous glands, their fibrous framework, and the deep position of their terminal saccules, enable them to energetically withstand inflammation, both chronic and acute, as well as new growths which have produced ulceration after infiltration of the connective tissue of the mucous membrane.

C. **Histological alterations of the mucous corium.**—We have already seen that, in consequence of catarrhal inflammation which has lasted some days, wandering lymph cells are found in the superficial elastic layer of the submucous tissue, as well as in the connective tissue which surrounds the blood-vessels, the excretory ducts, and the saccules of the glands. Bearing in mind what occurs in inflammation artificially induced in animals, either by subcutaneous injection of cantharidin or by irritating agents locally applied, the migration of lymph cells into the submucous fibro-elastic tissue must take place almost immediately after the commencement of the irritation. They are very numerous if the inflammation is acute, such as may be observed in the laryngo-tracheo-bronchitis of an eruptive or of typhoid fever. In the last named disease particularly a very marked infiltration of the submucous fibro-elastic layer may be observed; the lymph cells are arranged in a layer beneath the basement membrane and in the meshes of the network of superficial vessels which are distended with blood. These lesions are the cause of the ulceration of the larynx observed in typhoid fever. In acute inflammation, as in the laryngo-tracheo-bronchitis of variola, the whole of the submucous tissue is infiltrated with lymph cells, particularly the superficial fibro-elastic layer around the glands and blood-vessels.

In old chronic inflammation, such as is observed in tuberculosis, syphilis, lupus, lepra, bronchiectasis, &c., a more or less considerable thickening of the submucous tissue takes place; it is at first infiltrated with lymph cells, and often thickened in consequence of a new formation of bundles of connective tissue. In

the subacute or chronic form the following lesions of the connective tissue may be remarked: inequalities on the surface of the mucous membrane, superficial or deep ulcerations, and vegetations on the surface of the corium. It is rare to find ulceration on any part of the respiratory track unpreceded by a neoplasm which has a tendency to break down into caseous degeneration; inflammation, however, when it is acute, particularly if in relation with infectious microbia, as is the case in variola, diphtheria, glanders, typhoid fever, lepra, &c., may also lead to ulceration. When the ulcers undergo cure, fleshy granulations are produced; often, however, the work of repair started by this inflammation becomes excessive, and the fleshy granulations persist on the surface of the mucous membrane, or may even become organised and covered by a normal epithelial lining. On the other hand, we are certain that fleshy granulations may be developed on the surface of the mucous membrane of the air-passages without previous ulceration. Thus in certain forms of chronic bronchitis, such as that which accompanies dilatation of the bronchi (fig. 13), for example, projecting fleshy granulations are often found developed in the diseased bronchi. On microscopically examining vertical sections of the mucous membrane passing longitudinally through these granulations, a basement membrane of normal thickness, transparency, and homogeneity may be seen on their surface, under the epithelial investment (see also fig. 11).

D. **Lesions of the smooth muscles.**—In slight inflammation, the smooth muscles which surround the large and medium-sized bronchi do not undergo change. In acute inflammation they become infiltrated with lymph cells, which, lodged between the muscle cells, prevent these exercising their function, and as the result the bronchus dilates, which dilatation often persists for some time after recovery from the bronchitis. If the muscle fibres have been compressed by a large number of lymph cells effused into the muscular tissue, and if the inflammation continues for some time, they atrophy and are partially destroyed, which partial destruction may result in permanent bronchial dilatation. The anatomical cause of permanent bronchial dilatation is more or less complete atrophy of the muscle fibres of the bronchi, accompanied with the destruction of part of the elastic fibres of the fibrous coat. Acute inflammation invading the whole of the bronchial wall is generally related to the presence of tubercle in the lung. The bronchial tube, modified in structure and having

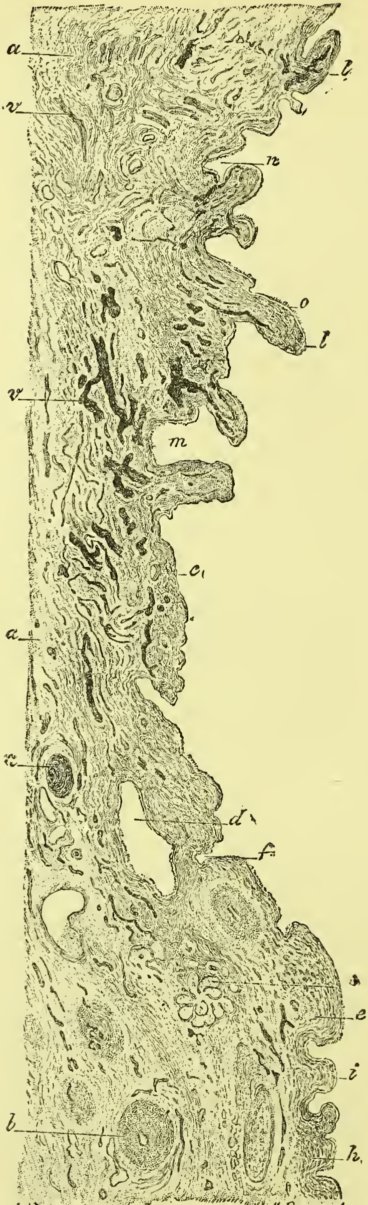


FIG. 13.—LONGITUDINAL SECTION OF THE WALL OF A DILATED BRONCHUS.

The internal surface of the bronchus, *c*, is everywhere lined with the basement membrane, *i*, on which may be seen some epithelial cells, which appear, under the low power used, as small points; *l, l*, projecting fleshy granulations; *f*, piles separating them; *v, v*, blood-vessels; *s*, a gland. Magnified 20 diameters.

lost partly or entirely the elastic and resistant elements that compose it, becomes dilated by the accumulation of a muco-purulent secretion, by inspiration, and by the efforts of coughing. The muscular fasciculi of the membranous part of the trachea and those of the large bronchi are often invaded by tubercles.

E. Alterations of the cartilages of the air-passages.—Morbid changes in the cartilages of the larynx, trachea, and bronchi are very common in deep inflammation, due either to an acute process, as in typhoid fever, or to a chronic process, as in tuberculosis and syphilis. The perichondrium then becomes infiltrated with a considerable number of lymph cells, which may be numerous enough to form a layer of pus around the cartilage, which becomes necrosed if completely isolated in the midst of the pus ; but more frequently it is subsequently inflamed. The cartilage capsules of the periphery, which are thin and elongated parallel to the surface, become larger, oval and spherical, at the same time that their cells proliferate and secrete new capsules around themselves. This new formation of cells takes place equally in the central part of the cartilage, where normally the cells are globular. Where it is very active the proliferation ends in the formation of small round or embryonic cells, which fill the primary capsules. Blood-vessels from the neighbouring tissue penetrate into these cavities, and on their internal surface a calcareous border is developed and imprisons the cells, which subsequently become bone-corpuscles ; in fact, the inflamed cartilage is changed into bone provided with large medullary cavities and bony trabeculæ, exactly as in the physiological ossification of cartilage (see vol. i. p. 368, fig. 15). Ossification of the cartilages is very frequently observed in all chronic inflammation of the air-passages, not only in the larynx but also in the trachea and bronchi. These newly-formed bone-plates may also become necrosed if bathed in pus, or be eliminated by way of the air-passages from the efforts of coughing, or from the surface of the skin, when, for example, an abscess developed round the cartilages of the larynx opens externally. All the lesions of cartilage cells already described (vol. i. p. 376) may be met with in the cartilages of the air-passages.

II. General Pathological Histology of the Lung.

The ultimate terminations of the bronchi and pulmonary alveoli, which are the essential parts of the lung, present for study an

epithelial lining and a framework of elastic and connective-tissue fibres. We will thus examine successively the histological changes of the epithelium and of the fibrous tissue of this organ before considering these lesions from the most general point of view.

Changes in the pulmonary epithelium.—In the embryo the epithelium completely fills the pulmonary alveoli. In the fœtus at term, who has not yet breathed, this epithelium is composed of a layer of large cells which have the form of truncated pyramids, the base of the pyramid being in relation with the wall of the alveoli. When respiration commences the air enters the lung, dilates the alveoli, and the cells which line their walls being intimately connected together gradually flatten to allow the movement of expansion. They first become cubical and finally quite flat, as in the adult, when the new-born infant has breathed for some time. Also in new-born children attacked during the first days of life with pulmonary congestion or broncho-pneumonia, and who have hitherto only breathed very incompletely, a condition will be found which is called by pathologists the fœtal state or broncho-pneumonia of new-born infants, in which the pulmonary alveoli will be found to be lined with a layer of large cubical or pyramidal cells, similar to those of the lung of the fœtus. Similar lesions may be observed when sections of syphilitic gummata of the lung in new-born infants are examined under the microscope. The alveoli, contained in the new tissue of the gumma, not having been dilated by the pressure of the air, their cells still preserve the characters of the fœtal state (see later 'Gummata of the Lung'). Finally, the first phenomenon observed in every pulmonary inflammation, from whatever cause, is swelling of the protoplasm of the flat cells of the epithelium of the alveoli, and their return to an irregular polyhedral or cubical shape, similar to that found in the fœtus.

As we proceed to examine in detail the changes which take place in the epithelial cells in diseases of the lung we shall treat at the same time of the various exudations and the cellular elements which accumulate in the interior of the alveoli.

Artificial inflammation of the lung.—In order to study the initial phenomena of inflammation in the lung it is necessary to produce pneumonia experimentally in animals. One or two hours after having injected cantharidin under the skin of a rabbit, in the way already described (see vol. ii. p. 9), certain lobules or lobes are generally found congested on the surface of the lung,

where the pulmonary tissue appears under the visceral pleura to be redder than normal. In sections made through these lobes, after hardening and staining with picrocarminate, the smallest bronchi may be seen to be almost completely filled with cubical or irregular, polyhedral, epithelial cells and with lymph cells. It may be supposed that the lymph cells are derived from the blood-vessels of the submucous connective tissue, for the latter contains vessels between its fibres. On the inner surface of the pulmonary alveoli is seen a layer of large granular polyhedral cells, containing one ovoid nucleus lying against its wall, between the capillaries gorged with blood, which project in a marked manner. Many of these large cells are free in the interior of the alveolus, where they are mixed with lymph cells.

If a small cubical portion of the congested lung of a rabbit which has been poisoned by cantharidin, be removed and placed, without compressing it, in a 1 per cent. solution of osmic acid, the elements are perfectly fixed in the position which they occupied during life. In this way the space occupied by the air in the bronchi and alveoli of the lungs can be estimated. In sections examined with a medium power (sixty to eighty diameters) it will be seen that the smallest bronchi and the alveoli have centrally a cavity half as small as in the normal condition, and that their walls appear to be much thicker. This cavity, which is quite circular, represents the air bubble which it contained at the moment that the fragment was placed in the osmic acid. The reagent has coagulated the fluid exuded into the alveolar and bronchial walls, and it has fixed the lymph and epithelial cells found there, so that these elements seem to coalesce with the wall. With a higher power the parts composing the exudation attached to the alveolar walls may be easily made out, and the tumefied epithelial cells, the lymph cells, and the red blood corpuscles can be recognised. The largest of the bronchi also show an exudation containing lymph cells, cylindrical cells, and red blood corpuscles, which exudation forms a thick layer attached to the wall, and is limited towards the centre of the bronchus by festoons which indicate the form of the air bubble which it had contained. In the same sections, tumefied endothelial cells may be seen on the inner surface of the blood-vessels, the protoplasm and oval nucleus of which project in a marked manner into the vascular lumen, and also lymph cells forming an adherent layer. These phenomena point to irritation of the internal membrane of

the vessels and explain the facility by which diapedesis takes place.

Similar forms of pneumonia, more or less acute, can be produced artificially in animals by injecting irritating substances, either in solution or suspended in a fluid, into the pulmonary vessels by means of the jugular vein, or into the bronchi, or into the pulmonary tissue itself. If, for example, a cubic centimetre of a 1 per cent. solution of nitrate of silver be injected into the lung of a rabbit, by means of a syringe introduced through the pectoral wall at an intercostal space, the fluid penetrates successively into a pulmonary lobule, a bronchus which opens into it, and thence into the neighbouring lobules and larger bronchi. The nitrate of silver will even irritate the trachea, and a spumous mucus may be seen to flow from the nose of the animal. If the animal be killed an hour after the injection, a part of a lobe of the lung will be seen, with the naked eye, to be tumefied and œdematous. If sections made after hardening in alcohol be examined under the microscope, the internal wall of the pulmonary alveoli will be found to be almost entirely denuded, with the exception of a few tumefied and granular cells. In the cavity of the alveoli a few spherical and granular cells, evidently derived from the epithelium, will be found as well as a small number of lymph cells, red blood corpuscles, and filaments of fibrin. This experiment shows how rapidly the lesions of pneumonia are produced. Twelve hours after the injection of nitrate of silver the pulmonary alveoli are filled with lymph cells contained in a close network of fibrillar fibrin. The state of the lung is then exactly similar to that of the lung of a man in acute lobar or fibrinous pneumonia. From these examples it is seen that the first phenomena of pulmonary inflammation experimentally produced consist in swelling of the epithelial cells, which very often show proliferation of their nuclei, a granular condition and division of their protoplasm, in desquamation of this epithelium, and in extravasation of the white and the red blood corpuscles, and of the liquor sanguinis, more or less rich in fibrin.

The inflammation runs a course varying with the special action of each irritant agent. If the dose of cantharidin is not fatal, the accidents caused by this substance in the lung and the bronchi ought to cease at the end of from twelve to fifteen hours. The epithelial cells of the alveoli should then be reapplied against the alveolar wall, though they still remain slightly tumefied. In pneumonia caused by the injection of nitrate of silver, hepatisa-

tion—that is to say, distension of the alveoli by lymph cells and fibrin—will last some days. The cells then become filled with fat granules before being taken up and carried away by the lymph vessels. If the pneumonia is the result of the injection of solid substances suspended in fluid, and of which the lung frees itself with difficulty, resolution is slow, and, particularly in the rabbit, the cells extravasated into the alveoli become infiltrated with fat granules; a condition of hepatisation is then produced which, to the naked eye, appears like caseous pneumonia. The evolution of pneumonia is, again, much slower if it has been caused by in-

FIG. 14.—ALVEOLI OF THE LUNG IN PULMONARY CONGESTION.

The wall, *e*, of the alveolus shows the blood-vessels, *d*, filled with red corpuscles, *g*. Along the wall at *h* may be seen flat cells, either detached or adherent. One of them to the left of the figure contains three nuclei. The contents of the alveolus consist of fine trabeculæ of fibrin, *a*, enclosing some lymph cells, *b*, and red corpuscles, *c*. Magnified 250 diameters.

fectious irritant agents. Thus, for example, if a small amount of pus derived from the sputa of a phthisical patient be injected into the jugular vein of a rabbit, a caseous lobular pneumonia is produced consecutive to the presence of pus cells in the capillaries of the lung.

If the two recurrent laryngeal nerves be divided in the rabbit, the consequent paralysis of the glottis facilitates the entrance of foreign bodies, such as the buccal mucus and fragments of alimentary matter, into the larynx and bronchi. A slow and insidious bronchitis and pneumonia is soon produced. Some weeks after the operation a more or less complete lobar or lobular hepatisation

will be found at the autopsy. The surface of a section of the diseased parts shows a yellowish or grey colour, resembling that of caseous hepatisation in man. The pulmonary alveoli contain a large number of epitheloid cells, such as may always be found in catarrhal pneumonia of slow progress. The yellow colour of the nodules of subacute catarrhal pneumonia is constantly observed in the rabbit, whatever may be the cause of the pneumonia, and must not be mistaken for a sign of tuberculosis. If, instead of dividing the recurrent laryngeals, the trunk of the two pneumogastric nerves be cut in the neck, the œsophageal nerves as well as the laryngeal nerves are paralysed, so that food and foreign bodies

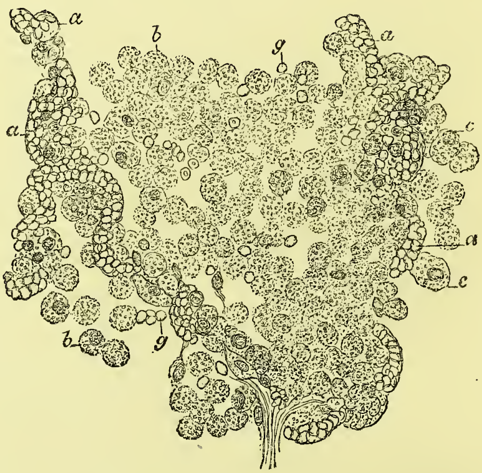


Fig. 15.—Catarrhal Pneumonia in the acute stage.

a, a, projecting vessels of the alveolar septa, filled with red blood corpuscles; *b, b*, lymph cells extravasated into the alveoli; *g, g*, red blood corpuscles, also free in the alveoli. Along the wall of these cavities are seen tumefied epithelial cells, some adherent and others partly detached, as at *c*. Magnified 250 diameters.

penetrate more easily into the lung; the pneumonia becomes then more acute and rapidly proves fatal. We shall consider later on the pneumonia caused in children and adults by foreign bodies, and which is comparable to pneumonia produced artificially by division of the pneumogastric nerve.

Inflammation of the lung in man.—The changes which take place in the cells of the alveoli in inflammation observed in man are similar to those already described in experimentally induced pneumonia. Whenever active congestion of the lung is present the epithelial cells become tumefied, their protoplasm granular,

and the liquor sanguinis is effused together with some red corpuscles and lymph cells into the cavity of the alveoli. If the inflammation continues, the epithelial cells proliferate at the same time that the lymph cells are extravasated in still larger numbers from the blood-vessels into the alveoli. In the acute period of inflammation the cavity of the alveolus is completely filled with these cellular elements situated in the midst of the exuded fluid, which latter often contains filaments of fibrin. Later on these cells become filled with fat granules, atrophy, and are absorbed by the lymph vessels. When the inflammation is declining, and diapedesis of the white blood corpuscles is less active, there will be found at the periphery of the alveolus a layer of large

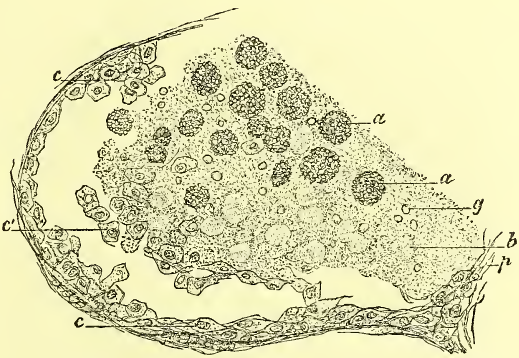


FIG. 16.—ACUTE FIBRINOUS PNEUMONIA IN THE STAGE OF RESOLUTION.

c, c, cubic, polyhedral, or flat cells applying themselves against the wall of the alveolus, *p*; *c'*, similar cells which are adhering to the intra-alveolar exudation. In the granular exudation contained in the alveolus, which has been coagulated by osmic acid, may be seen large round cells, *a*, filled with fat granules, and clear spaces, *b*, which have contained the preceding elements, but they have been detached in the course of preparation. *g, g*, red blood corpuscles. Magnified 200 diameters.

tumefied granular cells with one large oval nucleus, which cells adhere together or show a tendency to apply themselves against the alveolar wall and to form at a certain time a new epithelial membrane. This formation of an alveolar lining composed of large cells is found in the course of slight subacute catarrhal pneumonia, in the stage of resolution of acute pneumonia (see fig. 16), and in chronic pneumonia, whether catarrhal, fibrinous, or interstitial, accompanying pulmonary tuberculosis and new growths of the lung (see fig. 17, *b*, *b*). It is probable that these tumefied cells are simply lymph cells which have become hypertrophied by assimilating the substances contained in the fluid exuded into the alveolus. They are arranged along the wall of

the alveolus, adhere together, then become flattened against the alveolar wall, and finally form a lining similar to that which existed in the normal condition.

To recapitulate: at the commencement of inflammation the cells of the alveoli tumefy, proliferate, become globular, spherical, or polyhedral with blunt angles, and fall into the alveolar cavity, where they are mixed with lymph cells. The latter are the more numerous the more acute the inflammation. In subacute inflammation, such as catarrhal pneumonia and pneumonia undergoing resolution, and in the fibrinous pneumonia of tubercle, the large

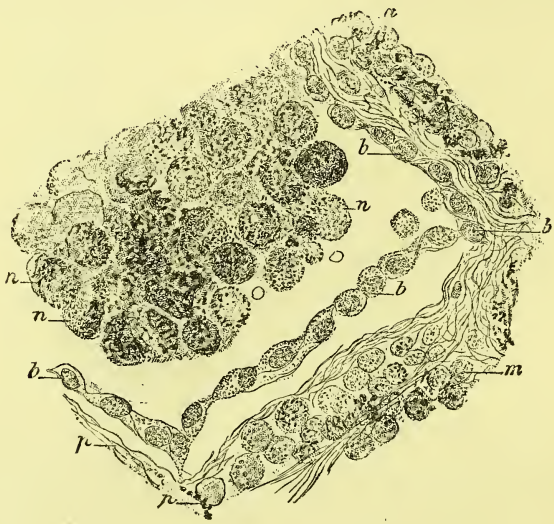


FIG. 17.—INTERSTITIAL CHRONIC PNEUMONIA.

a, thickened wall of the alveoli; along this wall, from *a* to *p*, may be seen flat or oval cells, *b*, *b*, adhering together and forming a kind of membrane applied against the wall or detached at places. The contents of the alveolus consist of large round and pigmented cells, *n*, *n*. Magnified 300 diameters.

epithelial cells are, on the contrary, as abundant and even more numerous than the lymph cells. Finally, the same large cells arrange themselves at the end of the inflammatory process along the wall of the alveoli and reconstitute their epithelium.

Fatty degeneration of the pulmonary epithelium.—In guinea pigs poisoned by arsenious acid or by phosphorus, we have found the epithelial cells of the alveoli tumefied, globular, filled with large drops of fat, and in a condition of fatty degeneration. Many of these modified cells become free in the interior of the alveolus,

the internal surface of which is denuded. These lesions of the epithelium coincide with pulmonary congestion and apoplexy.

Lesions of the connective tissue of the lung.—The fibrous framework, which circumscribes the lobules and surrounds the bronchial tubes and the blood-vessels, and which forms the septa of the alveoli, often undergoes changes in various diseases of the lung. Though these lesions are sometimes observed in chronic bronchopneumonia, they are more frequently met with in a peculiar form of chronic pneumonia, in chronic pleurisy, pulmonary tuberculosis, anthracosis, tumours, &c. They will be described with each of these various diseases.

The lesions of the lung hitherto described, whether the changes in the cells or in the connective framework be considered, greatly resemble those of connective tissue in general and of the serous membranes. The pulmonary alveolus may, in fact, be looked upon as the equivalent of a mesh of connective tissue or of a small serous cavity.

CHAPTER III.

SPECIAL PATHOLOGICAL HISTOLOGY OF THE RESPIRATORY SYSTEM.

I. Lesions of the Nasal Fossæ.

Congestion, hæmorrhage.—Congestion of the pituitary membrane is observed as the initial phenomenon of coryza or of epistaxis. It may depend on diseases of the heart or lungs, tumours of the nasal fossæ, or of certain general febrile diseases, such as typhoid fever, measles, &c. Hæmorrhage or epistaxis is sometimes primary, and often occurs in young subjects, and sometimes secondary, as in typhoid fever, hæmophilia, scrofula, acute fevers, &c. It is not known if these various forms of hæmorrhage are dependent on lesions of the walls of the vessels, or if they are solely due to an increase of blood tension.

Inflammation of the mucous membrane of the nasal fossæ, coryza.—Acute coryza begins by congestion accompanied with a serous exudation. The first drops of fluid which flow from the nasal fossæ already contain lymph cells, though it is quite transparent. The presence of these elements in the exudation of coryza quite at its commencement show that they are white blood corpuscles extravasated from the vessels and not the products of multiplication of the epithelial cells. Multiplication and the changes of the epithelial cells in this fluid are studied the more easily the more closely the secretion is observed. The cylindrical cells become globular and are sometimes segmented, so that a spherical element, having the diameter and shape of a pus corpuscle, shows, by still possessing cilia, from whence it is derived (see 5, fig. 18). They are distinguishable from the lymph cells with which they are mixed by the fact that they do not contain glycogenic matter, while the latter are more or less filled with it. When these elements are present in great numbers, the secreted fluid becomes turbid and may be even puriform.

Acute secondary coryza is observed in the exanthematous fevers, particularly in measles, erysipelas of the face, diphtheria of the pharynx or larynx, and in glanders, when these various diseases spread to the nasal fossæ. Each one of these affections is manifested in the mucous membrane of the nasal fossæ by certain local characteristics and by a course peculiar to itself. Thus in erysipelas, the mucous membrane and the submucous tissue are swollen and œdematous; in variola, pustules are formed, and in diphtheria, the false membrane characteristic of this disease. In glanders, the pituitary membrane is of a bright red colour, covered

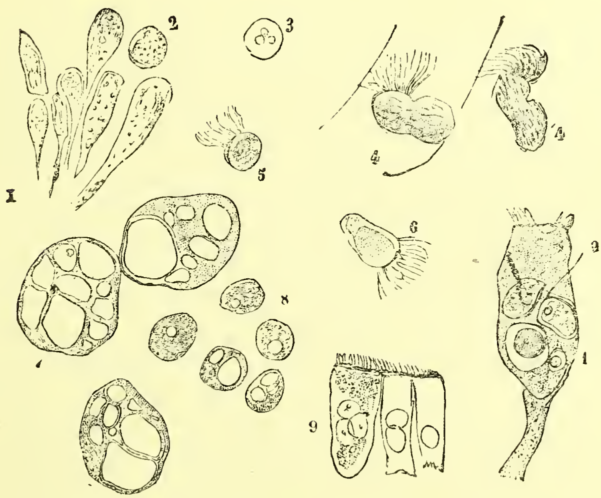


FIG. 18.—CELLULAR ELEMENTS IN THE MUCUS OF CORYZA.

1, detached cylindrical cells full of mucus; 2, 3, pus cells; 4, detached cylindrical cells with a cilia in movement; 5, portion of a cylindrical cell resembling a pus cell, but with its cilia preserved; 6, cylindrical cell which has become globular and preserved its cilia; 7, large globular cells containing drops of mucus; 8, lymph cells; 9, 9, cylindrical cells containing many nuclei. Magnified 500 diameters.

by a purulent exudation which is followed by small miliary, deeply placed abscesses, isolated or confluent, and by ulcerations. Finally, in this disease there may be consecutive inflammatory lesion of the periosteum and the bones. Acute inflammation is either cured or becomes chronic. Frequent and chronic coryza causes a thickening of the connective tissue of the mucous membrane and of the submucous tissue, which it is considered may be the point of departure of polypi of the nasal fossæ. It is also accompanied with ulcerations and the formation of small abscesses in the submucous tissue; it is rare for the periosteum and the bones them-

selves not to be attacked with ostitis or even to become necrosed. Chronic coryza is generally only observed in scrofula or syphilis. Deep lesions of the mucous membrane and of the submucous tissue, ulcerating gummata, necrosis of the bones which form the framework of the nose and of the palatine arch, cause perforations of this arch and of the soft palate, exfoliation of fragments of bone, flattening of the nose, &c.

Tumours of the nasal fossæ. Mucous polypi.—Mucous polypi of the nasal fossæ are generally developed in the mucous membrane of the interior of these cavities; they may be either solitary or multiple. Their pedicle is more or less thick, and their form corresponds more or less to that of the cavity, their size varying from that of a pea to a nut. They are soft, of a mucoid appearance, tremulous, and easily torn; they generally project towards the anterior nares, but in exceptional cases they project from the posterior nares; they take origin in the connective tissue of the mucous membrane and in the submucous tissue. On examining a delicate section of one of these tumours it may be seen that it is limited by a layer of cylindrical, stratified, ciliated epithelium, which belongs to the mucous membrane. In some of them the mucous glands may be seen to open on their surface; these glands are sometimes greatly hypertrophied, and their excretory ducts and acini present cystic dilatations similar to those found in mucous polypi of the uterus (see vol. i. p. 290). The cystic tumours of the maxillary sinus described by Giraldès belong to this variety. In certain mucous polypi of the nasal fossæ no glands are met with. The mucous tissue which forms the mass of the tumour is highly vascular, and contains bundles or fibrils of connective tissue separated by a mucoid fluid and by connective-tissue cells. A rather curious fact observed in all tumours of the nasal fossæ is that when they project externally the mucous membrane of the extruded part is lined with a stratified pavement epithelium.

Fibrous polypi.—The fibrous polypi of the nasal fossæ, as well as those which spring from the palatine arch in front of the basilar process and from the inferior surface of the body of the sphenoid, take their origin from the periosteum and even from the bone itself. The fibrous polypi of the nasal fossæ generally spring from the posterior part of these cavities; they send prolongations in all directions, into all the cavities, overcoming or avoiding all obstacles in their way, enlarging the nasal fossæ,

thinning and destroying the bones, and penetrating either by new passages or by natural openings, across the spheno-palatine foramina for example, into the sinuses which surround the nasal fossæ and into the zygomatic fossæ. They are composed of fibrous tissue, and, according to Muron, they contain a considerable number of capillary vessels with delicate embryonic walls, and it is this which gives them their hæmorrhagic character. Sarcomata may have the same seat and the same cause. It is probable that many of the tumours described under the name of fibrous polypi of the nasal fossæ are in reality sarcomata.

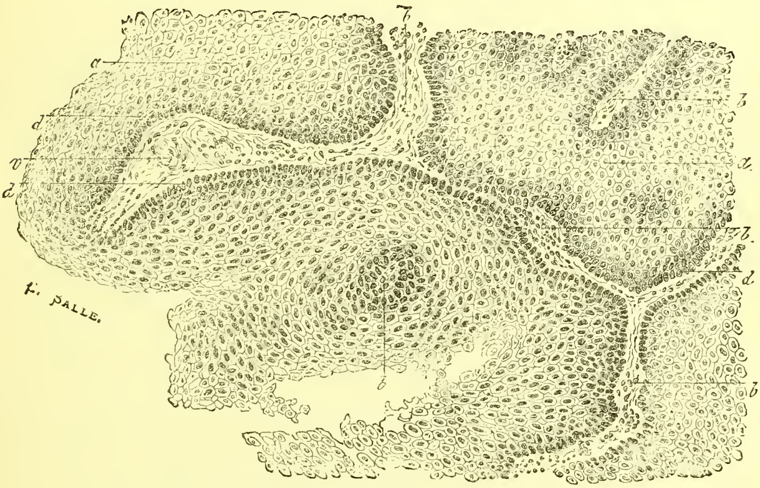


FIG. 19.—PAPILLARY POLYPUS OF THE NASAL FOSSÆ.

a, pavement epithelial cells, the deepest situated of which, *d*, are cylindrical and implanted on the papillæ, *b*, which latter contain blood-vessels, *v*. Magnified 80 diameters.

Primary carcinoma of the nasal fossæ is very rare; we have, in fact, never known of an authentic case.

Papillary polypi.—Among polypi of the nasal fossæ there are some which are truly papillomata. They are formed of numerous composite papillæ pressed one against the other, or contained within a common epithelial investment. Their stroma is fibrous and vascular, slight in quantity, while the epithelial investment is thick and composed of pavement cells (see fig. 19).

Pavement-celled epithelioma may be primarily developed in the lining of the vestibule of the nasal fossæ, or it may be caused by extension of epithelioma developed in the skin of the nose, the cheek, the upper eyelid, the alæ of the nares, or the upper

lip. In the nasal fossæ may also be found a form of cylindrical-celled epithelioma which is developed primarily in this situation, and which cannot be differentiated with the naked eye from mucous polypi (see vol. i. p. 277). In the maxillary sinus may also be observed polypoid tumours, which often extend into the nasal fossæ, and which belong to the order of tubular epithelioma. These tumours rest on a basis of morbid tissue, they are highly malignant, and their stroma is very rich in blood-vessels and embryonic tissue.

II. Lesions of the Larynx.

Congestion of the larynx, and acute catarrhal laryngitis.—Acute catarrh is primary, caused most frequently by chill, or it is secondary, accompanying a febrile exanthema (scarlatina, measles, &c.) Congestive and inflammatory redness, as well as swelling of the laryngeal mucous membrane, may be observed during the life of patients by laryngoscopic examination; but after death the mucous membrane becomes less red, owing to the fact that the blood is pressed out of the small blood-vessels. The phenomena which occur in the inflamed mucous membrane are the same as those already described when treating of artificially induced inflammation, and inflammation of the air-passages generally considered (see vol. ii. p. 9). It consists in stasis of the blood current and extravasation of the lymph cells from the vessels into the submucous tissue, and from there to the base of the epithelial lining. The cells of this lining are separated and crowded together by the passage of the lymph cells; they soon undergo more or less change, become desquamated in places, and an irregular lining is formed. At the same time an exudation of a mucous fluid may be observed on the surface of the mucous membrane; this is at first transparent, and contains globes of mucus, a few caliciform or cylindrical cells and lymph cells, but later it becomes thicker in proportion as the latter elements are more abundant; this fluid is expelled by coughing. The sputa expelled in laryngitis contain a few cylindrical cells still furnished with cilia. In slight laryngitis these cells generally persist beneath the layer of mucous or puriform exudation; these cells may nevertheless be found in the different states which indicate proliferation (see figs. 18 and 19). In certain cases of subacute irritation of the mucous membrane, the lesion consists essentially in a mucous state of the cylindrical cells, a great

number of which have become goblet-shaped. In a section of the epithelial lining, large mucous cells may be seen with clear contents and without cilia, the nucleus of which is pushed towards the base of implantation on the basement membrane. These goblet cells alternate with thin cylindrical cells flattened by pressure, and which possess a tuft of fibrillar cilia (vol. ii. p. 12, figs. 6, 7, 8). This state of the cells proves their active participation in the secretion of mucus. On the surface of the mucous membrane there are balls of mucus, debris of caliciform cells and a few lymph cells contained in the mucous fluid. In this case there are generally no lymph cells at the base of the mucous lining, for the inflammation is slight; if the inflammation is acute, the submucous tissue is infiltrated with lymph cells, particularly its superficial layer and around the blood-vessels. This lesion of the connective tissue is particularly well marked at the level of the aryteno-epiglottic folds in the laryngitis of measles. The glands of the laryngeal mucous membrane secrete a great part of the mucous exudation. Their saccules increase in size, the cells which they contain become tumefied, and the lumen of

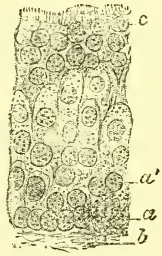


Fig. 20.—Section of the Mucous Membrane of the Trachea of a Rabbit, in Artificial Inflammation caused by Cantharidin. Section made after hardening the piece in Osmic Acid.

b, surface of the submucous tissue; *a*, round cells situated at the base of the epithelial lining; *a'*, the same cells occupying almost the entire extent of the section of the epithelial lining; *c*, layer of round cells furnished with cilia. Magnified 250 diameters.

the saccules and excretory ducts is filled by a mucous fluid which contains an abundance of lymph cells and caliciform cells. This condition corresponds with a marked hypertrophy of the glands visible to the naked eye, and on pressing the mucous membrane a small drop of muco-pus can be squeezed from each orifice (vol. ii. p. 18).

Chronic catarrhal laryngitis.—This disease is either the sequela of acute catarrh or the consequence of chronic granular pharyn-

gitis, of tuberculosis, or some other disease of the larynx. The mucous membrane is congested, brown or grey, and secretes a mucous or puriform fluid; it is thickened and its glands are hypertrophied. Hence this affection has been called *glandular angina*, a name which is applicable only when the glands of the pharynx are hypertrophied in a similar manner. The hypertrophy of the glands sometimes causes distinct elevations on the mucous membrane. In fact the layer of hypertrophied glands in front of the arytenoid cartilages may become from 3 to 5 mm. thick. In inflammation of long duration the connective tissue may itself undergo more or less considerable hypertrophy, and give origin to more or less numerous growths and papillæ. These growths, which are often limited to the vocal cords, or some other region of the larynx, constitute small sessile or pedunculated tumours. These lesions are accompanied by changes in the epithelium, which from cylindrical becomes stratified pavement. Fœrster was the first to describe this condition under the name of *dermoid metamorphosis of the laryngeal mucous membrane.*

Diphtheritic laryngitis, or croup.—Diphtheritic laryngitis is sometimes primary, sometimes caused by the extension of the disease developed primarily in the pharynx or in the lower parts of the air-passages. It is particularly observed in children. It begins by an inflammation purely catarrhal in character, but the mucous membrane soon shows false membranes. These are whitish or grey, varying in extent and thickness, and arranged in superimposed layers, the deeper layers of which are developed in contact with the mucous membrane, while the most superficial become disintegrated and are thrown off. They are more or less resistant, sometimes thick and difficult to tear or to detach, sometimes, on the other hand, soft and broken down into a caseous or granular detritus. These differences in consistence depend solely on the age of the false membrane. It sometimes happens that at the autopsy of children, who have expectorated large quantities of thick false membrane, scarcely anything can be found in the trachea or larynx except a slight pulpy detritus. In vol. i. p. 104, we have already described this peculiarity in describing the changes in the epithelium in diphtheritic inflammation. The false membrane, expectorated by coughing, has the form of that part of the mucous membrane which it covered; for example, if derived from the trachea or the large bronchi it may have a tubular appearance and even show bifurcations. The false mem-

brane expelled during the life of the patient is seen to be composed of fibrin in the form of filaments, of pus cells, and of epithelial cells. The latter, which are derived from the ciliated cylindrical epithelium or the pavement epithelium of the inferior vocal cords, are modified in their form and chemical composition, as has been already described in vol. i. Developed from the epithelial lining of the mucous membrane, the membranes are pushed forward as new cells are formed below them. They never contain blood-vessels. At the autopsy it may be seen that the mucous membrane, which is beneath the false membrane, is hyperæmic, red, infiltrated, and softened, and between the two there is often an

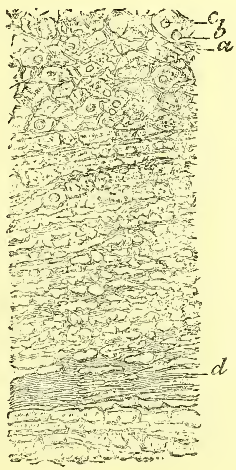


FIG. 21.—SECTION OF A DIPHTHERITIC FALSE MEMBRANE.

a, very fine fibrils of fibrin in the meshes of which are found lymph cells, *b*, and red blood corpuscles; at *d* the fibrils are thicker. Magnified 200 diameters.

infusion of blood, so that ecchymotic islets are observed on the inferior surface of the false membrane.

Sections cut perpendicularly to the surface of the mucous membrane of the larynx, covered by a diphtheritic false membrane, show the following details. On first examining the false membrane it may be seen that it is formed of fibrils of fibrin, composing a network of unequal meshes. Sometimes the fibrils of the fibrinous network are extremely fine, and the spaces between them are then generally rather large (fig. 21). In these meshes are lodged red blood corpuscles, lymph cells, or even a few tumefied epithelial cells. At other times the fibrinous trabeculæ are thick, close together, homogeneous, and refractile, and

the meshes between them are so small as to scarcely leave space to lodge, here or there, a red blood corpuscle or a lymph cell (fig. 22). The thicker the trabeculæ the smaller the spaces between them and the fewer the cellular elements. The fibrinous trabeculæ stain red with picrocarminate of ammonia.

It is between the meshes of this fibrinous network that is found, according to Klebs, the chief mass of the diphtheritic microsporon, the spores of which are often united into ball-like masses. Beneath the fibrinous false membrane, and between it and the basement membrane (fig. 23), are found lymph cells and deformed epithelial cells, which resemble in no way the cylindrical cells of the laryngeal cavity nor the tesselated lining of the vocal cords. These cells are globular or irregular; their protoplasm is refractile, vitreous, and stains an orange-yellow with picrocarmi-

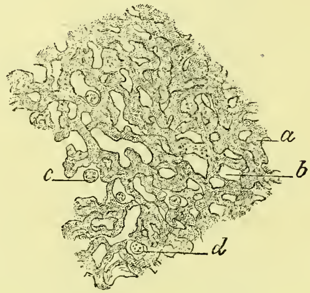


Fig. 22.—Section of a Diphtheritic False Membrane.

a, thick fibrils leaving small spaces, *b*, between them, in which are occasionally found a few lymph-cells. Magnified 200 diameters.

nate. Sometimes they form irregular masses, or a single layer situated between the false membrane and the basement membrane; at others they exist only as isolated cells, and the layer of fibrin adheres in places or for a great extent to the basement membrane, which is completely denuded of epithelial cells. The relation between the layers of fibrin and the morbid epithelial cells varies greatly. The latter, raised by the exudation thrown off from the surface of the submucous tissue, are arranged in layers which alternate with those of fibrin, or they are included in the fibrinous network. After a false membrane has been formed, globular epithelial cells are developed between it and the basement membrane, which raise and detach it gradually until it is expelled. The fibrin of which the false membrane is composed, is evidently derived from the blood contained in the blood-vessels of the subepithelial corium. It coagulates on the surface of the

latter by the same chemical process as on the surface of inflamed serous membranes (see vol. i. p. 104). The fibrino-plastic substance coagulates when acted upon by the fibrinogenic substance which is secreted either by the epithelial cells or by the red blood corpuscles and lymph cells extravasated from the blood-vessels together with the plasma. Beneath the fibrous false membrane is found the basement membrane, which is almost always preserved. It is, however, often creased and irregular. A few lymph cells may always be found adhering to the surface of the basement membrane and lodged in its folds. Sometimes the basement membrane disappears in very acute inflammation of the

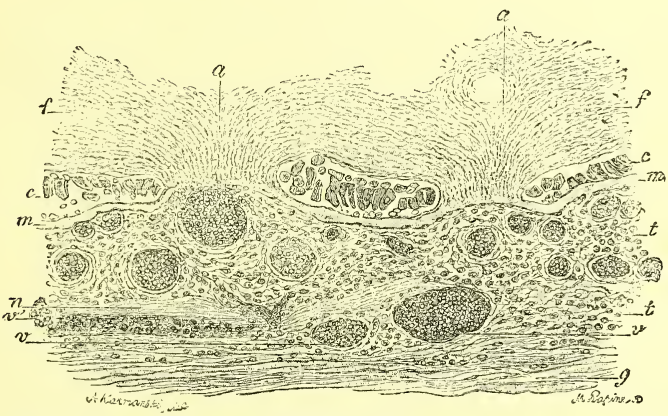


FIG. 23.—SECTION PASSING THROUGH THE BRONCHUS LINED WITH A FALSE MEMBRANE.

The false membrane, *f*, *f*, is composed of filaments and membranes of fibrin, which is attached at *a*, *a* to the mucous corium; *c*, *c*, cylindrical cells, more or less deformed and refractile; *m*, basement membrane; *t*, connective tissue of the mucous membrane, infiltrated with lymph cells; *v*, dilated vessels; *g*, muscular layer. Magnified 80 diameters.

submucous tissue. The superficial fibro-elastic layer of the corium and the submucous cellular layer are always infiltrated with a large number of lymph cells and red blood corpuscles; the serous exudation which is extravasated with the lymph cells between the fibres of the corium, may even contain fibrin coagulated in the form of fibrils. The superficial blood-vessels of the mucous membrane are very much dilated, filled with red blood corpuscles and a variable number of leucocytes; sometimes they are partially obliterated by a fibrinous thrombus; here also may be found spores and rod-like bodies. Very acute inflammation of the submucous tissue, accompanied with vascular dilatation and diapedesis, extravasation of the red blood corpuscles, and ecchy-

mosis, visible to the naked eye, extends deeply into the connective tissue as far as the muscles of the larynx. According to Klebs, the small rod-like bodies and spores of microsporon, are often found arranged in colonies, more numerous on the surface of the false membrane and throughout its whole thickness than in the submucous tissue. They diminish in number towards the deeper layers ; this arrangement indicates that they are derived from the air-passages. It is extremely rare to meet with true ulceration in laryngeal diphtheria. Laryngo-tracheal diphtheria may sometimes be found in the chronic condition, as has been proved by many cases published in the 'Bulletins de la Société anatomique.'

Erysipelatous laryngitis.—When erysipelas of the face or pharynx is propagated to the laryngeal mucous membrane it is characterised, as in the skin, by more or less marked redness and swelling of the submucous tissue. The cellular tissue of the mucous membrane is then affected with inflammatory œdema similar to that of cutaneous erysipelas and comparable to what is observed in œdema of the larynx. Suffocation, ending in death, may result.

Variolous laryngitis.—Inflammation of the mucous membrane of the larynx is very common in variola. E. Wagner, for instance, has in 160 post-mortems from variola observed laryngeal lesions in 144 cases. It must not be expected, however, that on opening the larynx pustules will be seen as well formed and as characteristic as those of the skin, or even of the buccal mucous membrane. Sometimes, however, we, as well as all other pathologists, have seen round elevations from one to two mm. in diameter, resembling pustules in shape and limited by a whitish friable cuticle, beneath which is a small drop of opaque fluid. Eruptions of this character simulate pustules of the skin. The eruption is often confluent and the mucous membrane is covered by a grumous pus or by more or less distinct pustules; at other times all that is seen is a very intense redness of the mucous membrane, which is covered by a muco-purulent secretion, in the midst of which may be seen opaque, white or yellowish-white grumous flakes, which are adherent to or partly detached from the epithelial layer. Often whitish exudations, attached or free, and which may be mistaken for the false membranes of diphtheria, are observed on the mucous membrane of the larynx when acutely inflamed. These false membranes may even become

blended into a thick layer lining a part or the whole of the larynx, even sometimes extending into the trachea. They may attain from half to one mm. in thickness. Beneath these different exudations the mucous membrane is inflamed, red, and sometimes ecchymotic, showing, for example, small red circular spots of the shape of ecchymotic pustules. The surface of the

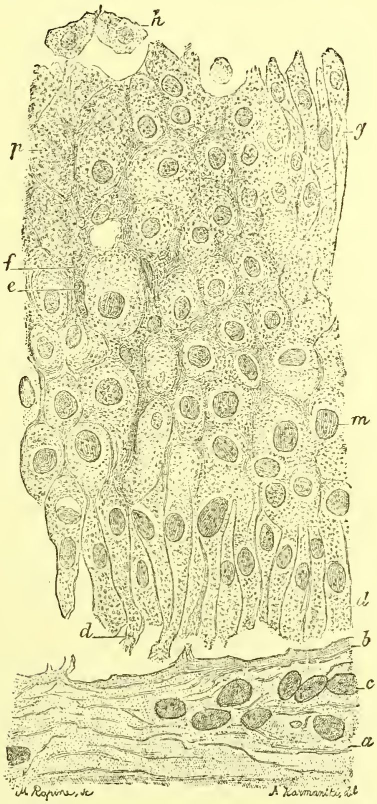


FIG. 24.—SECTION OF THE EPITHELIAL LINING OF THE LARYNX IN VARIOLOUS LARYNGITIS.

a, submucous connective tissue; *c*, lymph cells; *b*, basement membrane (the thickened epithelial lining extends from *d* to *h*); *d*, *d*, elongated cylindrical cells implanted on the basement membrane; *m*, lymph cells; *f*, protoplasm; *e*, nucleus of a flat cell; *h*, prismatic cell approaching the pavement shape; *g*, much elongated cylindrical cell. Magnified 350 diameters.

mucous membrane is often irregular, but it is not ulcerated. On section the submucous tissue is found to be considerably thickened and infiltrated with a red fluid.

Acute inflammation carried to its maximum degree may be studied in variolous laryngitis by means of the microscope.

From other points of view the lesions are variable. In microscopic sections of the mucous membrane, made through the epithelium where it is thick, the epithelial cells are seen to be much more numerous than in the normal condition; if, for example, a portion of the larynx which is lined with cylindrical cells be examined, eight to ten superimposed layers of these cells will be found. Round lymph cells derived from the corium will be seen in places to have hollowed spaces for themselves between the cylindrical cells, and two, three, or a larger number may be counted, placed one behind the other, in a small round or elongated space, the wall of which is formed of cylindrical cells (see this arrangement represented in fig. 5). Elsewhere cells which are round as normally, but much elongated and cylindrical will be found in contact with the basement membrane (fig. 24). The superficial cells have generally lost their cilia. In the grey or yellowish grey gruma or spots there are, according to Eppinger,[1] granular cells rendered cloudy by the micrococci which they contain; rows of these cells alternate with cells which are clear and less infiltrated. Everywhere, moreover, in the epithelial cells which are undergoing desquamation, in the lymph cells, in the mucus, and in the false membranes, which we are about to describe, disseminated spores are found, or conglomerations of micrococci.

At the point, where occur the prominent and whitish elevations which resemble pustules, and which may be regarded as the product of pseudo-membranous inflammation, the following superimposed layers may be observed in passing from the surface to the deeper parts; they are represented in fig. 25 :—

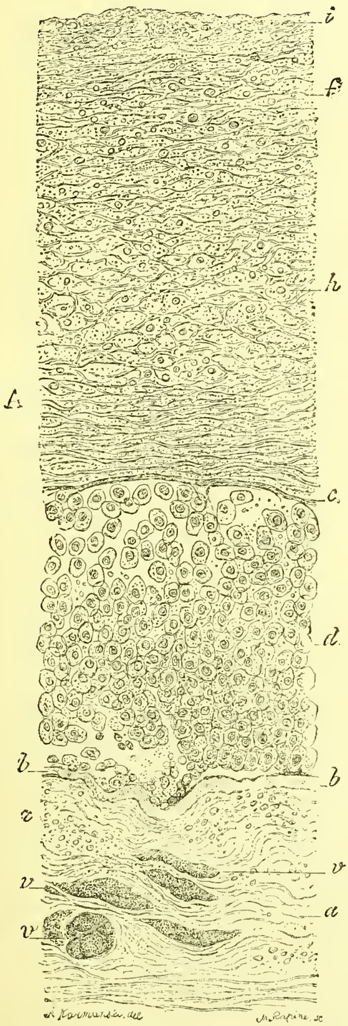
First, a fibrinous false membrane, composed of a network of filaments of fibrin. The meshes of this network are filled with lymph cells or red blood corpuscles, and moreover within the lymph cells which are on the trabeculæ of fibrin, or in the spaces which they limit, are seen granules which are supposed to be the spores of micrococci. These elements are shown at C, fig. 25, under a high power. The trabeculæ of fibrin become larger as the deeper layers are approached.

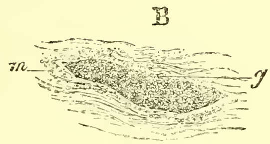
Secondly, a thick zone of epitheloid indifferent cells, larger than the lymph cells, and containing a large nucleus (*d*, fig. 25). These cells, arranged in many layers, are of an irregular polygonal form from reciprocal pressure. Among them also are found lymph cells.

Thirdly, an irregular and crumpled basement membrane

[1] *Handbuch der pathologischen Anatomie von Klebs,* vol. ii. p. 88 and following.

(*b*, fig. 25). Both below and above it are seen a large number of migratory cells, and at certain places it seems to show a solution of continuity to enable these elements to pass. Such are the phenomena which occur on the surface of the laryngeal mucous





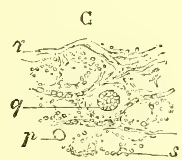


Fig. 25.—Section of the Tracheal Mucous Membrane at the Level of a Variolous Pseudo-membrane. Magnified 200 diameters.

A. The false membrane extends from *i* to *c*; in it may be observed filaments of fibrin, *f*, and lymph cells, *h*, contained in small lacunæ between the fibrils; *d*, layer of epitheloid indifferent cells; *b*, basement membrane of the mucous membrane; *v*, vessels filled with micrococci; *a*, elastic and connective-tissue fibres.

B. One of the vessels, *v*, with its contents magnified 350 diameters. *g*, contents of the vessels formed of micrococci, granules of which may be found in the surrounding connective tissue at *m*.

C. Superficial portion of the false membrane magnified 350 diameters. *r*, fibrils of fibrin; *p*, red blood corpuscle; *g*, lymph cell full of micrococci; *s*, the same micrococci granules free in the meshes of the fibrinous network.

membrane during the first days of a variolous eruption. At the same time the blood-vessels of the submucous tissue may be seen to be considerably distended, and this tissue becomes infiltrated with lymph cells and red blood corpuscles, escaped by diapedesis. At a certain moment stasis of the blood current occurs in

the capillaries, which are filled with micrococci and lymph cells, which latter contain masses of micrococci, as shown in fig. 25, B, *g*, under a high power. Inflammation of the submucous tissue is shown in the glands by changes in the acini as well as in the excretory ducts. The excretory ducts are filled with mucus, which contains lymph cells and mucous cells; this mucus spreads over the surface of the mucous membrane and mixes with the other products of exudation. The granular acini may contain granular indifferent cells as well as mucous cells, and the glandular connective tissue is completely infiltrated with lymph cells. This inflammation often extends to the borders of the cartilages.

When variolous inflammation of the larynx is acute its duration is rather long. At the autopsy of subjects who have died fifteen to twenty days after the commencement of the disease we have found the submucous tissue intensely inflamed; the mucous membrane, the surface of which was very red and covered with a puriform secretion, had totally lost its epithelium. Sections of this membrane showed, in fact, above the preserved basement membrane only a layer of small cubical cells—that is to say, lymph cells pressed one against the other. Laryngeal inflammation may terminate by a rapidly fatal œdema. It sometimes also causes perichondritis and deep abscesses.

Laryngitis of glanders.—We have had the opportunity of studying the lesions of the larynx in glanders in the human subject from their commencement. The laryngeal mucous membrane was found to be the seat of small whitish, prominent, and very superficial nodules about the size of a pin's head, from which a small drop of pus issued when they were pricked. On microscopic examination the pus was found to be collected between the epithelial lining and the submucous connective tissue. Groups of these small miliary abscesses had joined and produced ulcerations, the bases of which, impregnated with pus, were granulating and pultaceous (see vol. i. p. 211).

Laryngitis of typhoid fever.—The laryngitis of typhoid fever is more or less acute, and presents many distinct varieties for study.

a. In subjects who have died from pulmonary and bronchial complications, superficial catarrhal inflammation of the larynx is almost always found. In sections of the mucous membrane, the epithelial layers are found thicker and richer in lymph cells than

normally. The acinous glands contain a large quantity of mucus mixed with lymph cells, and the elastic and connective tissue between the basement membrane is very often seen to be infiltrated with many lymph cells, whilst the capillaries of the mucous membrane are dilated and filled with blood.

b. In a more acute form of laryngitis, and one more characteristic of typhoid fever, the lymph follicles are tumefied and form nodules similar to those formed by the isolated follicles of the small intestine in the same disease. In sections of these nodules the meshes of retiform tissue are seen to be plugged with lymph cells, some small, others larger, and containing two or three nuclei. This multiplication of the nuclei and this infiltration of the retiform tissue are entirely similar to what is observed in the closed follicles of the small intestine. The connective tissue surrounding these altered follicles is equally inflamed.

c. The nodular tumefication of the closed follicles is often followed by cupular or crateriform ulcerations, which have been well described by Louis. These ulcerations are usually seated, like the nodules already described, at the base of the epiglottis, on the internal surface of the arytenoid cartilages, and on the anterior commissure. They are hollowed, and rest on an indurated infiltrated base, with raised and hard edges. Similar ulcerations are often found at the same time on the mucous membrane of the posterior wall of the pharynx and at the root of the tongue. In microscopical examination of sections carried through these ulcerations, lymph cells may be seen on the point of becoming detached at the border of the loss of substance. The tissue of the mucous membrane which forms the border and base of the ulcer is infiltrated with numerous lymph cells, pressed one against the other.

d. In certain cases of typhoid fever, a pultaceous, yellowish layer, resembling a false membrane, may be found on the mucous membrane which lines the posterior surface of the epiglottis and the arytenoid ligaments. It is feebly adherent, and is composed of changed epithelium disintegrated and separated by masses of micrococci (Klebs). The epithelial cells are even sometimes so much flattened and compressed by the mass of parasites that they form trabeculæ between them. Sometimes the framework of the false membrane of this laryngitis is formed of filaments and laminæ of fibrin, as occurs in the false membrane of diphtheria. According to Klebs the micrococci belong to a form of microphytes, which become rod-shaped at a later period of development, the bacillus of typhus.

e. Klebs has described two cases of ulceration, in one of which the vocal cords and in the other the upper half of the epiglottis was destroyed, and in which the connective tissue was in a state of molecular disintegration, and the epithelium on the border of the lesion, and the connective tissue which formed its base were all infiltrated with large masses of micrococci. In these two cases the cartilages were also invaded by the microphytes, and in sections, lines of rod-like bacteria, either simple or jointed, were seen to protrude into the matrix of the cartilage. The cartilage cells were in a state of granulo-fatty degeneration, and were breaking down. Here was a case of gangrenous inflammation, caused by the presence of an infectious microbium.[1] Whatever may be the cause or the starting-point of laryngeal ulcerations in typhoid fever, if it causes perichondritis by extension to the deep layers of the mucous membrane it may produce very serious results—necrosis of the cartilages, purulent foci, and œdema of the larynx.

Syphilitic lesions of the larynx.—Syphilis shows itself in the larynx by catarrhal inflammation, mucous plaques, superficial and deep ulcers, and by all the accidents which result therefrom—

[1] In many cases which we have examined there was present, together with ulceration of the free edge of the epiglottis, an intense laryngo-trachitis, excited by the presence of a thick false membrane adherent to the mucous membrane of the internal surface of the epiglottis, the vocal cords, the ventricles, and the whole laryngeal cavity. In one of these cases the false membrane was continued into the small bronchi. Under the false membrane the mucous membrane was red, ecchymotic, and puffy. Sections cut perpendicular to the surface of the laryngeal mucous membrane at the level of the vocal cords showed in places, on examination, adherent false membranes which were composed, as in laryngeal diphtheritis, of filaments of fibrin, with which were entangled lymph cells and red blood corpuscles. Beneath the false membrane the basement membrane was seen to have disappeared at places where the inflammation was most intense. In the submucous tissue the vessels were largely dilated and filled with blood or plugged by fibrinous thrombi, and the connective tissue was infiltrated with red blood corpuscles, lymph cells, and sometimes also with fibrin. This inflammation extended deeply into the acinous glands and throughout all the connective tissue as far as the cartilages. The mucous membrane was thickened, so that the ventricles of the larynx were almost entirely effaced. In the laryngeal cavity the submucous tissue was less altered and preserved its basement membrane; its surface was, however, covered with false membranes, beneath which were found, in places, tumefied, deformed, vitreous, cylindrical cells, or globular cells, which stained orange-yellow by picrocarmin. The false membrane, composed essentially, as in true diphtheria, of fibrin, was attached to those parts of the basement membrane which were quite bare. On the surface of the false membrane and in its meshes very fine grains of micrococci were easily seen, and rod-shaped bodies sometimes jointed like bacteria.

perichondritis, œdema, &c. Mucous plaques are characterised by a slight elevation, and are produced chiefly by swelling and proliferation of the epithelial cells. All the deep syphilitic lesions of the mucous membrane cause proliferation and granulation of the connective tissue, which is generally more extensive than in diseases of the larynx due to other causes.

Tubercular lesions of the larynx.—The laryngitis of tubercle is characterised by various changes, simple catarrhal inflammation, tubercle, ulcerations, perichondritis, ossification of the cartilages, &c. (*vide* pp. 50, 55.)

Œdematous laryngitis, œdema of the glottis.—This form of laryngitis, which is sometimes primary, sometimes secondary to albuminuria, tuberculosis, glanders, syphilis, variola, typhoid fever, erysipelas, anasarca, extensive burns of the skin, &c., consists in a serous or puriform infiltration of the submucous connective tissue. The œdema is more often localised in the upper part of the larynx. The aryténo-epiglottic folds, which are swollen, œdematous, flaccid, and semitransparent, fall together, and, acting like a valve, close the larynx during inspiration, while the expired air can still pass by raising them. The arytenoid region is as œdematous as the base of the epiglottis, and all the other parts of the mucous membrane may be the seat of a similar swelling, for the œdema is very often caused by a local lesion, by ulceration which has extended to the cartilages, or by a deep purulent focus. The œdematous mucous membrane is red, livid, or pink; on incising it a marked quantity of serous or puriform fluid flows out spontaneously or on pressure. On microscopical examination of fragments of this œdematous tissue, removed by scissors, connective-tissue bundles may be observed separated from one another, as in œdema of the connective tissue, by a more or less transparent fluid containing lymph cells, and sometimes a reticulum of fibrin. In this fluid may also be found swollen and granular connective-tissue cells and red blood corpuscles. When the lesion is consecutive to deeply-seated changes in the larynx, such as ulcers or perichondritis, the lymph cells are much more numerous than in œdema which is primary or consecutive to Bright's disease.

Ulcerative laryngitis.—Ulcers of the larynx vary much. We have already seen that small ulcers may be developed in the laryngeal mucous membrane in acute primary laryngitis. In

typhoid fever, the ulcers are deeper, cup-shaped, and generally filled with a caseous detritus, and probably take their origin from an inflamed lymph follicle. The ulcers consecutive to **variolous pustules** are not so deep, but are more or less extensive, and result from the total destruction of the epithelial lining and partial destruction of the basement membrane. The exposed submucous tissue is congested, granulating, and covered with pus. In **tertiary syphilis**, ulcers are formed in the laryngeal mucous membrane, the base of which is granulating, while the submucous tissue is thickened, indurated, and highly vascular. These ulcers may extend over a considerable portion of the laryngeal mucous membrane, and even to the trachea. When seated on the epiglottis they destroy it, commencing from its free border. They often give origin to fresh granulations. These ulcers may be cured by proper treatment. The embryonic connective tissue develops into adult tissue, and the epithelial lining is reconstituted; but, owing to shrinking of the cicatricial connective tissue, narrowing of the larynx or trachea may result. The ulcerations of **glanders** seated in the larynx show the same tendency as in the nasal fossæ to destroy the tissues by phlegmonous suppuration.

Tuberculosis is much the most frequent cause of laryngeal ulcerations. They are met with particularly in pulmonary phthisis, which is coincident with a laryngo-bronchitis, and they generally extend into the trachea and the first divisions of the large bronchi. The laryngeal mucous membrane is then found to be very much congested and covered with a muco-pus, in the parts not ulcerated as well as at the level of ulceration. These ulcers are generally consecutive to tubercular granulations, either discrete or confluent, primarily developed in the submucous tissue. The form and arrangement of the ulcers are determined by the tubercular granulations themselves. Superficial at the commencement, they attack successively all the layers of the connective tissue of the mucous membrane; they may also be seen along the gland-ducts and in the glands themselves (*vide* p. 55). When the superficial tubercles become caseous, and all the mucous membrane is infiltrated and crowded with small cells, and the blood-vessels are obliterated, partial gangrene is produced, the first effect of which is desquamation of the epithelium and destruction of the basement membrane. A small circular erosion, $\frac{1}{2}$ to 1 mm. in diameter, soon appears, and gradually extends in width and depth by the destruction of the connective tissue infiltrated with caseous elements. This ulcer has tortuous, festooned borders, and each of

the granulations, or each of the groups formed by them, becomes the centre of a focus of destruction and consecutive eliminative suppuration. In consequence of the extension of the ulceration, flakes of thickened submucous tissue may be detached either from the surface or from the edge of the ulcers; the base of the ulcer is often fungoid, and is composed of vascular granulations which secrete a rather considerable quantity of pus. The inferior vocal cords at their point of insertion, the cords themselves, the arytenoid cartilages, the epiglottis, and the ventricles are the parts of the larynx which are most frequently the seat of these ulcerations. The exposed fibrous tissue of the vocal cords is itself sometimes eroded; the free edge of the epiglottis also often shows a loss of substance in which the cartilage is destroyed after proliferation of its cells. The submucous connective tissue is often very much thickened by the new formation of embryonal cells around and beneath the ulcerations; sometimes it is infiltrated with a more or less purulent serum. These lesions cause immobility and loss of function of the different parts of the larynx in which they are seated. The muscles are also sometimes attacked; the intermuscular connective tissue contains a more or less considerable quantity of embryonic cells and pus corpuscles, and the primary fasciculi undergo fatty degeneration. The cartilages, with the exception of the epiglottis, where the inflammatory lesions are, however, very frequent, are most frequently encrusted with calcareous salts, and they may even ossify. We will recur to this subject when treating of perichondritis. Deep ulceration or abscess of the larynx may end in perforation, the pus making a passage across the subcutaneous connective tissue to the surface of the skin in front of the neck, or across the walls of the œsophagus.

Perichondritis.—Suppurative inflammation of the perichondrium of the cartilages of the larynx may, according to certain published cases, be developed in consequence of an acute simple laryngitis caused by cold; but it is more often the result of one of the causes of ulcerative laryngitis and follows deeply-seated ulceration. In laryngitis which is deeply seated and of long duration, and in which the cellular tissue of the mucous membrane is infiltrated with lymph cells, the perichondrium is changed in the same way, and the cartilages themselves undergo modifications of nutrition. Sometimes calcareous infiltration may be observed in the matrix and the capsules; sometimes an actual ossification

is produced, which occurs, as in physiological ossification of the cartilages, by proliferation of the cells, dissolution of the capsules, and penetration of blood-vessels; cavities filled with embryonal medulla are thus formed, and bone corpuscles soon appear. These lesions of the cartilages are common in laryngeal phthisis. Slow perichondritis, which accompanies the ossification of cartilage, may be followed by suppuration of the perichondrium. The pus insinuates itself and separates the perichondrium from the cartilage, which mortifies by being isolated from its nutritive membrane. In cartilages necrosed in consequence of typhoid fever we have observed the matrix become granular, and the cartilage cells contain fat granules. The abscess, in the midst of which the cartilage is found, penetrates into the submucous tissue, and into the joints, and ends by protruding either under the laryngeal mucous membrane, or towards the skin, or into the pharynx. Before long it opens and emits, with the pus, fragments of cartilage, which are generally calcified or ossified, particularly if the case is one of laryngeal phthisis. The cartilages the most frequently affected are the thyroid, the cricoid, and the arytenoid. In the two first cases either the larynx or the skin may become perforated, from which results inflammatory subcutaneous œdema, and sometimes emphysema. When the arytenoid cartilage is diseased perforation generally occurs in the pharynx; laryngoscopic examination renders the diagnosis of this disease easy during the life of the patient.

Tumours of the larynx. Myxoma.—Small polypi, composed of mucous tissue, covered by a delicate mucous membrane, are occasionally found in the larynx. They are met with on the posterior surface of the epiglottis and in the ventricles of Morgagni.

Fibromata, or fibrous polypi, of the larynx are more frequent; they are generally small, varying in size from that of a mustard-seed to a pea. They slowly increase in size, as may be seen by laryngoscopic examination made at intervals of one or two years. They are sessile or pedunculated, and are most frequently seated on the inferior vocal cords. They are hard, resisting under the scalpel, and their tissue presents all the characters of fibrous tissue. Their surface is smooth or unequal, and covered by a stratified pavement epithelium. This epithelium is always of the same character, whether the polypus is developed on the vocal cord which is lined with pavement cells, or on the other parts of

the laryngeal mucous membrane, which are lined with cylindrical cells. These polypi sometimes undergo superficial ulceration. They develop primarily from the connective tissue of the mucous membrane.

Tubercle of the larynx.—If, before opening a tuberculous larynx, the upper part be examined, the free edge of the epiglottis may be often found tumefied and thickened, either regularly or in an unequal manner, presenting irregular depressions, even though the mucous membrane covering it shows no sign of ulceration. An erosion, or loss of substance, may also often be found on the free edge of the epiglottis, limited by the cartilage, which is itself partly destroyed or eroded. Similar lesions are found on the

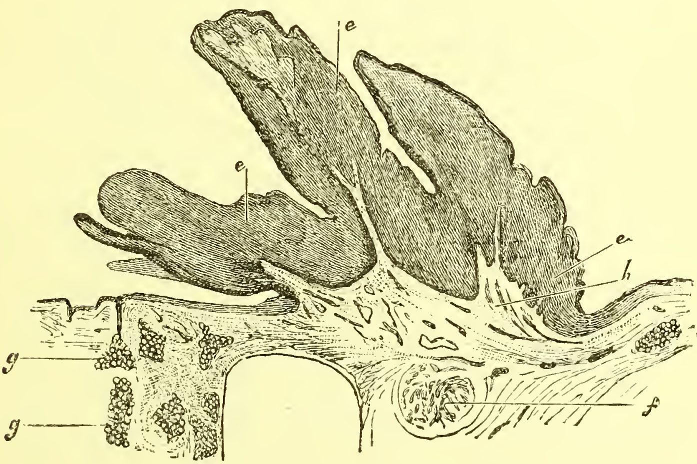


FIG. 26.—PAPILLOMA OF THE LARYNX.

e, *e*, papillæ; *h*, cellulo-vascular tissue; *c*, epithelial investment; *g*, racemose glands. Magnified 12 diameters.

superior and inferior vocal cords. A deep ulceration may sometimes be found on the cords, or on one only, situated either at the point of insertion or towards the middle, so deep, in fact, that the fibro-elastic tissue may be laid bare, or the cord may be detached, or partly or entirely divided. On opening the larynx, the mucous membrane of the ventricles looks swollen, fungoid, and covered with pus; small superficial, isolated, or serpiginous ulcerations are observed on the thyroid and cricoid cartilages; the mucous membrane is everywhere thickened and covered with muco-pus, which issues from the orifices of the acinous glands. Such is the usual appearance of the larynx in chronic tubercular laryngitis, but

tubercular granulations visible to the naked eye can rarely be observed; at the most, small greyish or yellowish slightly raised spots are all that can be discovered at the edge of the erosions. In recent miliary tuberculosis of the lung, on the other hand, slightly raised, flattened granulations are often observed on the laryngeal mucous membrane; they are very small, greyish or yellowish in colour, isolated or in groups, and are either covered by the mucous membrane or are undergoing ulceration. These granulations, and the serpiginous festooned granulations which succeed them, are especially characteristic at the level of the thyroid and cricoid cartilages.

To make a microscopic examination of tubercle of the larynx, and the various accompanying lesions, the pieces should be hardened by the successive action of Müller's fluid, gum, and alcohol, and sections should be cut perpendicularly to the surface of the mucous membrane. In such sections the tubercular granulations will very often be found at the spots where the mucous membrane is not ulcerated at all, or where it is simply irregularly tumefied, without showing projections or granulations visible to the naked eye. The thickened epiglottis, for example, shows first a stratified pavement epithelium in every way normal; beneath, the basement membrane is intact, but the superficial part of the submucous tissue is infiltrated with a great number of lymph cells, and from place to place are seen collections of these cells, in the midst of which one or more giant cells are formed. In the parts where the submucous tissue is inflamed the blood-vessels are dilated, filled with red blood corpuscles, containing also often an abnormal number of white cells; they are obliterated, and sometimes the presence of giant cells inside them may be ascertained, especially when they occupy the centre of tubercles. At the level of the vocal cords, and in the other regions of the laryngeal mucous membrane, the tubercles show the same peculiarities of structure. When a section passes through a tubercle which has undergone superficial ulceration, or through a mucous membrane infiltrated with tuberculous tissue, there is desquamation of the epithelial cells and the basement membrane disappears. The base of the little ulceration is formed of the tissue of the mucous membrane infiltrated with small granular cells, in the midst of which giant cells may also be observed. These latter, as well as the small cells, become gradually disintegrated, and some of them may often be seen on the point of becoming detached from the tubercular mass. When the mucous membrane is invaded

throughout its whole thickness, the ulceration extends downwards to the perichondrium, which is itself frequently inflamed.

The different layers of the connective tissue of the mucous membrane and the acinous glands are often affected throughout before any ulceration appears. The mucous membrane then appears as simply thickened, and its surface is a little irregular, but in the majority of cases no tubercular granulation can be distinguished with the naked eye. The tubercles are not only located in the stroma of the mucous membrane; they are also

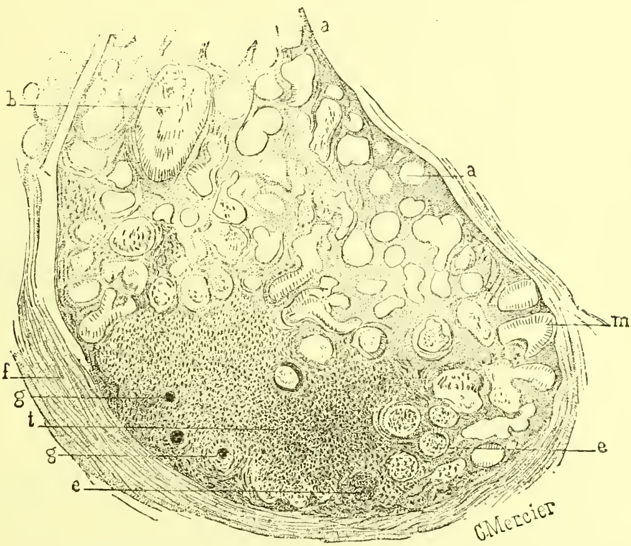


FIG. 27.—TUBERCLE DEVELOPED IN AN ACINOUS GLAND OF THE TRACHEA.

a, *m*, normal saccules; *b*, section of a rather large glandular duct; *t*, tubercular mass, in the midst of which giant cells, *g*, *g*, may be seen; *e*, *e*, saccules in which the epithelium and the contents are modified by inflammation; *f*, peri-acinous connective tissue. Magnified 20 diameters.

found at the rim of the glands, and in their acini. Thus a tubercular granulation may often be seen encircling the excretory duct of an acinous gland, particularly at its point of bifurcation, or even in the centre of a glandular lobule in the midst of normal or altered saccules (*vide* fig. 27). Elsewhere a tubercular granulation may be found astride as it were, one limb across the connective tissue which surrounds the gland, and the other in the midst of the glandular acinus. The tubercles in the glands are developed simultaneously from the connective tissue and from the epithelial cells within the saccules. In fact, in the substance

of the tubercular nodules may be seen the trabeculæ of connective tissue which separate the saccules, thickened and filled with lymph cells. Inside the saccules also the same elements may be found, mixed with mucous cells or epithelial cells with granular protoplasm. The latter, which result from the transformation of caliciform cells, no longer contain mucus, are polyhedric from reciprocal pressure, and have one large nucleus (*vide* fig. 28, B). A saccule may be sometimes entirely filled with these cells. In other parts of the granulation the trabeculæ of the connective tissue are no longer visible, owing to their being infiltrated with small round cells or giant cells. Giant cells may also be met with in a

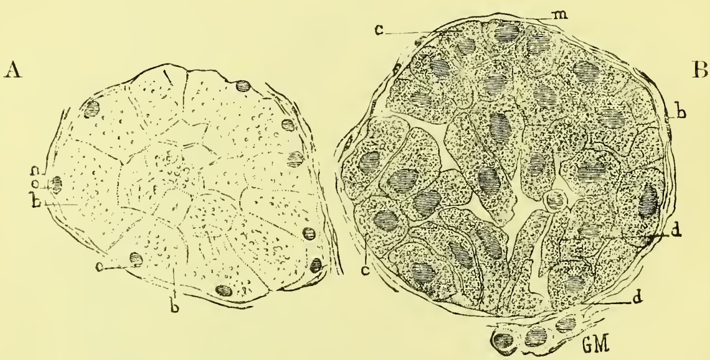


FIG. 28.—A. NORMAL SACCULE.

b, *b*, mucous cells with transparent contents, including refractive granules; *o*, small ovoid nucleus adherent to the proper wall of the saccule. Magnified 300 diameters.

B. GLANDULAR SACCULE FROM THE MIDDLE OF A TUBERCLE. SHOWS PROFOUND ALTERATION OF THE CELLS.

c, *c*, polyhedric or prismatic cells implanted on the hyalin wall, *m*, of the saccule, *d*; the cells are flattened from pressure. All these cells, which entirely fill the saccule, are large, irregular, and contain an opaque granular protoplasm and a large ovoid nucleus. Magnified 300 diameters.

saccule the limit of which is still well defined. Some of these cells are so large that each may of itself entirely fill a saccule. Some of them anastomose sometimes by means of processes with neighbouring giant cells, and aid in forming a network in the meshes of which small cells are found (*vide* fig. 29). Though a mucous gland may be thus compromised by a tubercular neoplasm, yet, if not entirely destroyed, the normal glands, or those simply inflamed, do not cease to secrete a large quantity of mucus. The excretory ducts of the glands also, even when surrounded by tubercular tissue, are more dilated than narrowed, and the mucous membrane being much thicker than normally, the ducts passing

through it become elongated. These ducts are lined with a layer of long cylindrical cells, which are mostly caliciform, and their lumen is filled with mucus, the debris of mucous and epithelial cells, and with pus cells.

All the lesions above described may be present without the occurrence of ulceration. When the mucous membrane ulcerates, the superficial layers of the submucous tissue, in which the tubercles are situated, become gradually disintegrated after desquamation of the epithelium and destruction of the base-

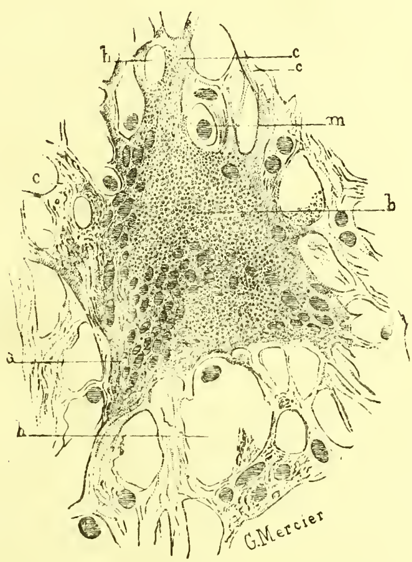


Fig. 29.—Giant Cell from the Tubercle of an Acinous Gland.

b, central granular mass of the giant cell; *a*, its numerous and superficial nuclei; *c*, its processes anastomosing in a network towards the periphery; *m*, cells contained in the peripheral network. Magnified 300 diameters.

ment membrane. An erosion is then produced, which is superficially bathed in a puriform fluid. At the same time that the tubercular masses are undergoing destruction the inflamed parts surrounding them give origin to fleshy granulations. The inflammation often extends downwards, and the connective tissue surrounding the cartilages becomes completely infiltrated with lymph cells; hence ensue inflammation and ossification of the cartilages. That part of the excretory ducts of the glands which is situated in the superficial layer of the mucous membrane is destroyed

when the submucous tissue is ulcerated, but the glandular saccules, which are more deeply seated, are perfectly preserved even when located in the midst of tissue infiltrated with lymph cells. The solid fibrous framework of these saccules offers great resistance to inflammatory and destructive processes.

Ecchondroses and **osteomata** have been observed on the internal surface of the cricoid cartilage.

Carcinoma.—Carcinoma of the larynx is not so rare as was thought some years ago. Blanc, Fauvel, Krishaber, and others have published a rather considerable number of cases. Independently of tumours which originate in the pharynx, and which afterwards invade the larynx, two varieties of primary tumours of this organ may be distinguished, according to their seat. In the first, the tumour takes origin in the cavity of the larynx itself, either below or above the glottis; in the second, the neoplasm is developed in the epiglottis, at the superior orifice of the larynx, and in its arytenoid wall. Carcinomata of the second variety are the most frequent.

As may be ascertained by laryngoscopic observations of patients extending over several years, laryngeal carcinoma is slow in progress. It is characterised by granulations of a dark red colour, ecchymotic in appearance, or of a yellowish colour, which ulcerate, break down, and give origin to pus. If extirpated these granulations return very rapidly. On examination they show the typical structure of carcinoma. After a period varying from three to five years, or even more, during which the neoplasm has made very slow progress, it rapidly extends to a larger surface of the mucous membrane, the granulations become larger, and the respiration becomes so impeded that tracheotomy is rendered necessary. After death, infiltration is found, and a more or less considerable granulation of the mucous membrane of the aryteno-epiglottic folds, and of that lining the epiglottis and the arytenoid cartilages, particularly if the tumour is external to the larynx. If the tumour is developed primarily in the laryngeal cavity, the mucous membrane covering the upper and even the lower vocal cords, the ventricle, and the subglottic region are affected; the submucous tissue and all the deep cellular tissue are also invaded by the neoplasm. The granulations and the thickened mucous membrane are soft in consistency, and yield on section the milky juice characteristic of encephaloid carcinoma. The diseased cartilages may themselves become ossified or calcified; they are sometimes necrosed and fragmented in the midst of the

cancerous tissue. In a case reported by Krishaber part of the ossified cricoid cartilage fell into the left bronchus and caused death. Ultimately carcinoma of the larynx extends to the lymphatic glands, to the pharynx, œsophagus, and neighbouring organs.

Epithelioma.—The autopsies, in which the nature of the cancerous tumours of the larynx has been exactly determined by microscopic examination, have not been sufficiently numerous to enable one to estimate the relative frequency of carcinoma and epithelioma of the larynx. Lobulated pavement epithelioma, which is sometimes met with in the larynx in the form of ulcerated granulations or growths, and which affects the mucous membrane throughout its whole thickness, may be mistaken for carcinoma. It is seated generally in the cavity of the larynx, on the vocal cords, in the ventricle, or in the subglottic region.

Papillomata, or **papillary polypi,** of the larynx are, after tubercle, the most frequently observed. These tumours have a cauliflower arrangement, showing a crowd of primary and secondary buds, and they have a great tendency to grow and spread. Each one of the papillæ composing a tumour is formed of a connective-tissue and vascular axis and stratified pavement epithelium. The papillæ are often contained in a common epithelial investment. The usual seat of papilloma of the larynx is at the angle of junction of the inferior vocal cords, and on the cords themselves; but they are also found in the ventricles of Morgagni and on the mucous membrane of the ventricle of the larynx.

Adenoma.—We have already explained, in describing chronic catarrhal laryngitis, how the glands of the larynx hypertrophy and acquire sometimes a considerable size, making the mucous membrane from 3 to 5 mm. thick. We have here, properly speaking, small adenomata ; they may—rarely, it is true—become more prominent and pedunculated. The acini of these hypertrophied glands are larger than normally, but their epithelial cells preserve their physiological character. They are caliciform, that is to say, filled with mucus. Papillary excrescences may almost always be found on the surface of these tumours. As hypertrophied glands often exist at the base of papillomata, it results that polypi of the larynx are often mixed tumours, in which sometimes the papillary, sometimes the glandular form predominates.

Lymphadenomata have been frequently observed in the mucous membrane of the larynx, trachea, and bronchi, in the form of soft nodules, more or less large and flat. Sometimes they cause a

diffuse infiltration of the mucous membrane (Virchow). They are recognised by their peculiar structure, which is that of adenoid tissue (*vide* vol. i. p. 245).

III. Lesions of the Trachea.

Congestion and inflammation.—Catarrhal congestion and inflammation of the trachea are frequent; they are caused not only by changes of climate, but are present in eruptive fevers, in typhoid fever, and in most diseases of the larynx, bronchi, lungs, and heart. Congestion occurs at the commencement of every catarrhal inflammation. It is shown by dilatation of the superficial vessels of the submucous tissue and by a change in colour of the mucous membrane, which becomes of a more or less dark red tint. The mucous membrane then becomes covered by an aerated mucus, which may be either transparent or cloudy from the presence of lymph cells. The redness of the mucous membrane is more marked between the cartilaginous rings and in the membranous portion of the trachea; it is at the same places also that the orifices of the acinous glands are most easily seen, and their excretory ducts are found to contain a considerable quantity of mucus, which may be squeezed out by tension or pressure of the mucous membrane. On examining sections of an inflamed trachea under the microscope, lesions already described (vol. ii. p. 12) are found in the epithelial investment, the connective tissue, and the glands; these lesions are seen in their most typical form in acute or chronic tracheal inflammation. The pustules of variola at their different states, the intense superficial and deep inflammation which they cause, the false membrane covering them when they are confluent, show the same details of structure in the trachea as in the larynx (vol. ii. p. 42). If diphtheria originates in the larynx it often extends into the trachea.

Tubercle.—In subjects who have died of pulmonary tuberculosis, post-mortem examination will show that though the mucous membrane of the trachea may be simply tumefied and unequal, covered with muco-pus, and seeming, at first sight, to have neither characteristic ulcerations nor granulations, microscopic examination often reveals tubercular infiltration. The submucous connective tissue, and that which penetrates between the bundles of smooth muscle cells and dips into the glandular lobules, is found to contain numerous tubercular granulations. But most frequently the surface of the mucous membrane shows, either the relief of

discrete or agminated tubercles, situated beneath the epithelial investment, or erosions and ulcerations caused by the destruction of an isolated tubercle, or many grouped together to form a superficial patch. The tubercular ulcerations of the trachea, more frequent than those of the larynx, are, like the latter, serpiginous, and more or less numerous and extensive; they are limited by a whitish sinuous and festooned border. Superficial at first, their base shows a red or greyish, often granulating, surface; by extending downwards the cartilaginous mass may become exposed. The most common seat of tubercular ulceration is between the cartilaginous rings or in the membranous portion of the trachea, but the mucous membrane covering the cartilages is sometimes invaded. The entire trachea is often covered by an eruption of confluent and partly ulcerated granulations. The tubercles are most frequently closer together, the neoplastic infiltration of the mucous membrane deeper, and the ulcerations more numerous and larger as the lower part of the trachea, near the point of division, is reached.

In sections of a tubercular trachea may be seen the lesions of the epithelium which have been already described in treating of inflammation of the air-passages (*vide* vol. ii. p. 15) and of tubercle of the larynx (*vide* vol. ii. p. 54). The arrangement of the tubercles, the tubercular infiltration of the submucous tissue, and of the glands are the same as in the laryngeal mucous membrane. The fasciculi of smooth muscle cells between the cartilaginous rings and in the membranous portions are generally affected; the connective tissue separating the muscular fasciculi is filled with small cells, which accumulate in islets from place to place, and in the midst of these are found obliterated blood-vessels and giant cells. The connective tissue surrounding the cartilages is equally inflamed. The latter often show multiplication of their cells, and an actual medullisation with the formation of osseous trabeculæ. Cartilage, which has become thus ossified, sometimes calcified, may have its perichondrium peeled off and become free in a focus of pus. These foci sometimes extend into the peritracheal connective tissue, and often communicate with a focus of caseous suppuration occupying the centre of a lymphatic gland.

Perforations of the trachea are produced in consequence of ulcers of the œsophagus, abscesses developed in the neighbouring connective tissue, cancerous tumours of the lymphatic glands and the œsophagus, and aneurism of the arch of the aorta.

Carcinoma is never developed primarily in the trachea, but it is not very unusual to find secondary carcinomatous nodules, circular and more or less voluminous in size, in the cellular tissue of the tracheal mucous membrane.

Leucæmic tumours have been observed here also.

Sometimes in old persons the cartilages of the trachea are calcified or ossified, and show exostoses on their surface or at their edges; two or more rings may even be solidly united by a bony growth binding together the neighbouring edges.

IV. Lesions of the Bronchi.

Congestion, hæmorrhage.—Congestion of the bronchi precedes and accompanies inflammation of the air vesicles, and is present in almost all respiratory and cardiac diseases. It also occurs in many fevers, such as the eruptive fevers and typhoid. It is characterised by fulness of the blood-vessels and swelling of the mucous membrane; and it is rare for congestion, even passive, not to be accompanied by a secretion of mucus. The internal surface of the congested bronchi is of a dull red. However short a time the congestion has lasted, or whether it be active or passive, the mucus secreted on the surface of the bronchi contains red blood corpuscles and lymph cells, and the epithelium soon shows the lesions of catarrhal inflammation. Ecchymoses of the mucous membrane are met with in the febrile exanthemata, in typhoid fever, scurvy, and asphyxia. The bronchial mucus then contains a more or less considerable quantity of red blood corpuscles. Large hæmoptyses are, however, always caused either by bronchial ulcerations, by rupture of an aneurism of a branch of the pulmonary artery into a tubercular cavity, or they are connected with pulmonary apoplexy, gangrene, or rupture of an aortic aneurism.

Bronchitis.—Catarrhal inflammation of the bronchi, whether acute or chronic, is due to the same causes as that of the larynx, and shows, in a general way, the same histological changes (*vide* p. 36). Limited to the large bronchi, it is not dangerous, but when it invades the small bronchi it is often fatal, particularly in young children and in old persons. It is then generally complicated with lesions of the pulmonary parenchyma, congestion, lobular or catarrhal pneumonia, atalectasia, emphysema, and sometimes even with lobular gangrene.

Acute bronchitis of the small tubes affects simultaneously those

which are not more than 1 mm. in diameter, and which are furnished with a complete muscular coat and with glands, the intralobular bronchi, and the acinous bronchi. The lesions differ in these different tubes. In the medium-sized bronchi, the diameter of which is more than 1 mm., the mucous membrane is tumefied, its surface is of a bright red and covered with mucopus. The bronchi, of from 1 to 3 mm. in diameter, are often dilated; puriform mucus accumulates within them, the longitudinal folds on the surface of the mucous membrane are effaced, and the bronchial tube seems more rigid than normally. The liquid which bathes the surface of the bronchi contains a large number of lymph cells, mixed with desquamated cylindrical cells; the epithelial investment undergoes the various changes already

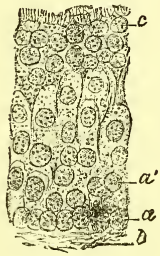


FIG. 30.—SECTION OF THE MUCOUS MEMBRANE OF THE TRACHEA, FROM A CASE OF INFLAMMATION INDUCED BY CANTHARIDIN. SECTION MADE AFTER HARDENING THE PIECE BY OSMIC ACID.

b, border of the corium; *a*, round cells situated at the base of the epithelial investment; *a'*, the same cells occupying almost the whole extent of the section of the epithelial investment; *c*, layer of round cells with vibratile cilia. Magnified 250 diameters.

described when treating of experimentally induced inflammation of the mucous membrane of the air-passages (*vide* p. 9); the lymph cells raise the layer of cylindrical cells, interpose themselves between them, and accumulate in the deep layers; the layer of ciliated cylindrical cells is irregular, and may even be wanting in places. In acute inflammation, the connective tissue of the mucous membrane is infiltrated with lymph cells, and these elements are also met with between the fibres which separate the bundles of smooth muscle cells; the sub-epithelial connective tissue becomes thickened in consequence, at the same time that the function of the smooth muscle cells is impeded. This is the principal cause of bronchial dilatation, to which may be added the accumulation of the fluid secreted and the efforts of inspiration. The acinous glands pour into the interior of the bronchi a

mucus containing epithelial cells which are more or less altered, and a rather large number of lymph cells. The saccules of the glands and their ducts, examined in sections, also show the presence of free lymph cells in their cavity or located between their epithelial cells. The blood-vessels are gorged with blood. Though the inflammation may affect the bronchus as far as its external coat, it shows, however, no tendency to invade the adjacent pulmonary lobules.

Capillary bronchitis.—But it is not so if the bronchitis attacks the intralobular bronchi, the calibre of which is less than 1 mm.

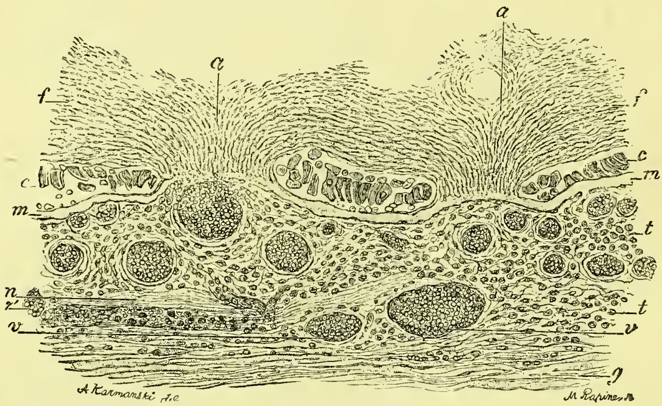


FIG. 31.—SECTION PASSING THROUGH A BRONCHUS LINED WITH A FALSE MEMBRANE.

The false membrane, *f*, *f*, is composed of fibrils of fibrin, which at *a*, *a* are implanted in the corium; *c*, *c*, the cylindrical cells are more or less deformed and refractile; *m*, basement membrane; *t*, connective tissue of the mucous membrane infiltrated with lymph cells; *v*, dilated blood-vessels; *g*, muscular layer. Magnified 80 diameters.

In these small bronchi there are no cartilaginous plates, and the muscular coat does not form a continuous layer, the contractile cells being simply disseminated in the midst of a connective tissue which surrounds the bronchus, with openings for the neighbouring alveoli. From the central bronchus of a lobule spring the terminal bronchi, which end in the acini. The intimate connections of the intralobular bronchus with the terminal bronchi and the pulmonary alveoli cause an entire lobule to almost always participate in inflammation of an intralobular bronchus. It is thus that lobular pneumonia is produced. As the terminal or acinous bronchi are affected in inflammation in the same way as the pulmonary alveoli, the description of their lesions will be given in the chapter on Broncho-pneumonia.

We shall now proceed to describe what occurs in inflammation of the intralobular bronchi. In sections properly hardened these bronchi will be seen to be almost completely filled with lymph cells, in the midst of which a few cylindrical cells are found. The layer of ciliated cylindrical cells is generally *in situ*; the connective tissue of the bronchial wall is uniformly infiltrated with lymph cells, the sub-epithelial layer as well as the middle layer which contains the smooth muscle cells, and the external layer which is in relation with the pulmonary alveoli. All the connective tissue which is around the small bronchus and the pulmonary arteriole which accompanies it, is inflamed in the same way. Hence it is that in a section cut perpendicularly to the

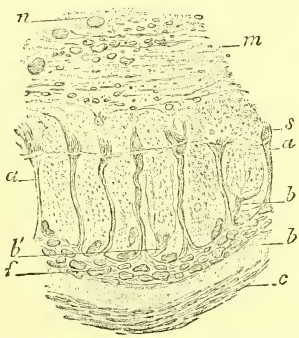


FIG. 32.—SECTION OF THE EPITHELIAL INVESTMENT OF A BRONCHUS OF MEDIUM SIZE AFFECTED WITH BRONCHITIS.

a, *a*, cylindrical cells flattened and compressed by the caliciform cells, *b*; *s*, vibratile cilia; *b*, *b′*, caliciform cells; *f*, round or slightly flattened cells at the base of the epithelial investment; *b*, basement membrane; *c*, smooth muscle cells; *m*, free mucous exudation on the surface of the epithelial layer; *n*, lymph cells and pellets of mucus. Magnified 300 diameters.

bronchus and the arteriole which accompanies it these tubes appear to be surrounded by a circular zone formed of connective tissue infiltrated with lymph cells. The lymph vessels which accompany the artery and bronchus are also filled with lymph cells.

Pseudo-membranous bronchitis is seen, especially in children, as a complication of croup; but it may also be observed in variola and laryngo-typhus, in adults as well as in children. It then generally coincides with pulmonary congestion, and with more or less extensive foci of broncho-pneumonia. The pseudo-membranes of the bronchi show exactly the same structure as those described in pseudo-membranous laryngitis.

Chronic bronchitis.—This form of bronchitis is very often

related to emphysema and cardiac diseases. Whether primary or secondary it is characterised by the greyish violet or slate-coloured tint of the surface of the mucous membrane. The connective tissue of the mucous membrane is frequently thickened by the formation of fibrous tissue, which often causes small superficial papillary excrescences. The mucus secreted is sometimes transparent, gelatinous, and small in quantity (the pearl-like sputa of Laennec); sometimes it is muco-purulent. At other times the expectoration is formed of a serous fluid which is exuded in great abundance (pituitary bronchitis, blennorrhagia of the bronchi). When the fluid secreted is mucous and gelatinous, the superficial cells of the investment are almost all caliciform and filled with mucus. These cells, distended with mucus, are lodged between the ciliated cylindrical cells (*vide* fig. 33).

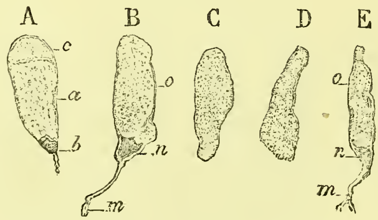


FIG. 33.—ISOLATED CALICIFORM CELLS.

In the three cells A, B, E the nuclei, *b*, *n*, *n*, may be distinguished, the protoplasm full of mucus, *a*, *o*, *o*, and the tail of the cells by which they are implanted on the basement membrane, *m*, *m*. The cell A is surmounted by a pellet of mucus, *c*. The two cells C and D are changed into a mass of mucus. Magnified 350 diameters.

Chronic bronchitis is sometimes accompanied with dilatation of the bronchi and ulceration of the mucous membrane. Tubercular bronchitis is described later (*vide* p. 73).

Dilatation of the bronchi. Bronchectasia.—Bronchi, dilated in consequence of an acute inflammation, return easily to their normal condition; but if the bronchitis passes into the chronic condition the bronchi often remain dilated. In chronic bronchitis, which is either simple or accompanied with pleurisy and chronic pneumonia, the bronchi often become permanently dilated; these dilatations are, however, produced more frequently in chronic tuberculosis. Sometimes the bronchi are uniformly dilated in their longitudinal direction; the dilatation is then said to be cylindrical. Sometimes such dilated bronchi form regular pouches near the pleura; at others there may be a succession

of cylindrical or ovoid dilatations along the course and divisions of a bronchus; this may be particularly observed in the apex of the lung; the dilated portions may be united by portions of bronchi of normal diameter. This is called moniliform or necklace-shaped dilatation (Cruveilhier). The third variety, which is the most common, consists in an ampullar or saccular dilatation, which is generally solitary and often of considerable size. When the ampullar dilatations are multiple, many of them intercommunicate by means of bronchi which are more or less dilated. A lung, or one of its lobes, may thus be converted into an alveolar cavity, the septa of which are formed of compressed and indurated pulmonary tissue. These dilatations are generally seated towards the periphery of the lung; they are then always accompanied by chronic pleurisy, characterised by fibrous thickening of the pleura. The bronchus, in its ultimate course and in its terminal ramifications, is atrophied beyond the dilatation, or some of its branches are transformed into cysts. More or less large cystic cavities are sometimes found in the apices of the lungs; they are lined with a mucous membrane and contain a mucous fluid. They are considered to be bronchial dilatations which have been isolated and closed after obliteration of the small bronchus which led to them. These lacunæ sometimes exist quite independently of any other bronchial dilatation. They must not be mistaken for lacunæ which are developed between the pleura and false fibrous membranes, due to chronic pleurisy. The walls of these pulmonary lacunæ are recognised by the arrangement of elastic fibres and the pigmentation of the blood-vessels found in them. The internal surface of those bronchial dilatations which still communicate with bronchi, and into which air still penetrates, is lined with a mucous membrane which is continuous with that of the normal bronchi. The mucous membrane of a bronchus which is completely dilated, and has at the same time ampullar dilatations—the most usual case—seems, on naked-eye examination, to be smooth, pink or grey, semitransparent, slimy, or covered with a transparent mucus. Beneath are seen the cartilaginous plates and the muscular fasciculi of the bronchus. These are no longer apparent at the level of the ampullæ, and are only seen distinctly on the surface of the less dilated bronchi which unite two ampullæ. The longitudinal fibro-elastic fasciculi are also less numerous and apparent; they are not seen in the ampullar portions of the bronchus.

If sections cut perpendicularly to the surface of the dilated

bronchi be examined under the microscope, the epithelial investment will be found in different states. The ciliated cylindrical cells are always preserved when the mucous membrane shows to the naked eye its usual polished surface. Chalice-cells very often exist in their proper place. When the dilated bronchus secretes a notable quantity of muco-pus, one or two layers of lymph cells will be found beneath the cylindrical epithelium in contact with the basement membrane. The cylindrical cells are then often desquamated in an irregular manner, in which case the epithelial investment is replaced by one or more layers of lymph cells; sometimes, however, the cylindrical cells are replaced by cubical cells without cilia, or by much elongated fusiform cells which end on the surface of the mucous membrane by a long process. Elsewhere are found small fusiform cells containing a nucleus in their swollen centre, and which are inserted by one of their extremities into the basement membrane, the other terminating in a blunt

FIG. 34.—SECTION OF THE SURFACE OF A BRONCHIAL MUCOUS MEMBRANE AT THE POINT OF DILATATION.

a, a slightly elongated cell midway between the cylindrical and cubical form; *d*, cell swollen centrally, implanted on the basement membrane by a delicate pedicle and terminating above by a blunt point; *m*, basement membrane; *b*, *c*, lymph cells situated in the submucous tissue. Magnified 200 diameters.

point (*d*, fig. 34). These cells irregularly edge the mucous surface. Beneath the basement membrane the connective tissue is rich in lymph and connective-tissue cells; its elastic fibres are more or less entirely destroyed. Sometimes on the surface of dilated bronchi the connective tissue forms vegetations or papillæ which are thinner at their bases than at their apices; they vary in number and size, and are visible to the naked eye. They are composed of fibres separated by connective-tissue and lymph cells; and they are bounded by the basement membrane and the epithelial investment which cover them entirely. Sometimes many of them are grouped together side by side, and between them, at their bases, are accumulated cylindrical cells full of mucus. The connective tissue which forms the framework of the bronchus, as well as that which accompanies the glands and supports the bundles of smooth muscle cells, is always thickened and more or less infiltrated with lymph cells. At the periphery of the bronchus it is continuous

with the pulmonary tissue, which at this spot is almost always affected with chronic inflammation. The mucous glands contained in the walls of a dilated bronchus are always more or less affected, and their saccules and ducts contain lymph cells and mucous cells. To study the changes in the muscular fasciculi, both longitudinal and transverse sections of bronchi affected with ampullar dilatations should be successively examined. These sections should be made in such a way that the longitudinal should pass through one or two ampullæ separated by contracted portions (fig. 35). Here two or three arcs of a circle are seen, the concave surfaces of which correspond to the ampullæ, which are separated by projecting ridges of bronchial mucous membrane. Generally no bundles of muscular fibres are found in the dilated portions of the ampullæ, but as the extremities of the arcs are reached a few isolated fasciculi are found cut across, and finally at the extreme ends of the arcs which represent the section of the ampullæ, namely at the points of sharp projection formed by the division of the contracted portion of the bronchus situated between two ampullæ, here the muscular fasciculi are preserved. Now it is

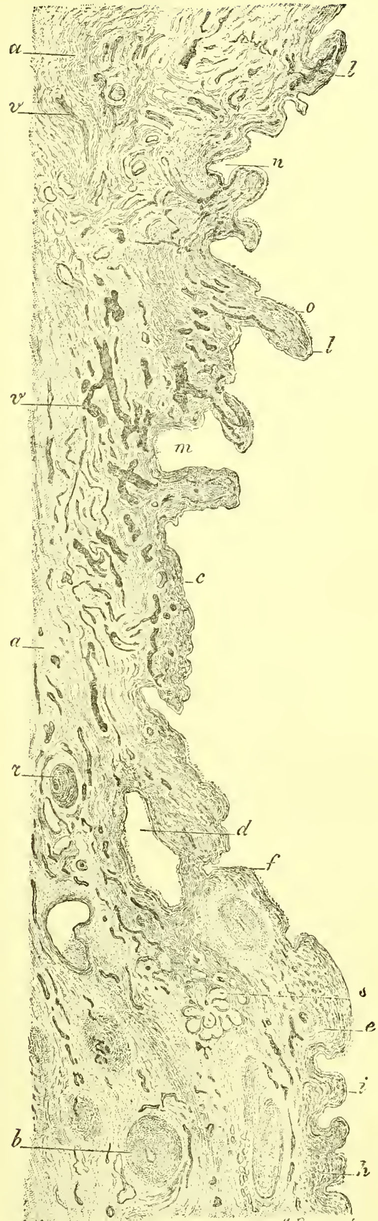


FIG. 35.—LONGITUDINAL SECTION OF A DILATED BRONCHUS.

The excavated portion of the bronchus from *l* to *e* represents an ampullar dilatation; *a*, connective tissue; *l*, *l*, prominent vegetations or papillæ on the internal surface of the bronchus; *e*, *h*, muscular fasciculi; *n*, *f*, interpapillary depression; *m*, deep depression limited by papillæ; *d*, depression of the same kind; *c*, surface of the dilated bronchus; here a basement membrane is seen which is present everywhere on the papillæ as well as in the depression; *v*, *v*, vessels filled with blood; *r*, artery affected with arteritis; *b*, section of another artery affected with endo-arteritis; *s*, an acinous gland. Magnified 20 diameters.

known that normally muscular fasciculi form a regular, uniform coat of equal thickness round a bronchus; hence it is seen that the muscular fasciculi are atrophied at the level of the ampullar dilatations of the bronchi. The projecting vegetations which have already been described are generally seen in the segment of the bronchus which separates the ampullæ; on the internal surface of the ampullæ similar vegetations are rarely seen. The fibrous coat of the bronchus is thinner at the level of the ampullar dilatations than in the contracted portions. It shows an uniform structure throughout its whole thickness, and is formed of parallel fasciculi of connective tissue separated by beds of flat cells; it is bounded on the side of the mucous membrane by a basement membrane and an epithelial investment, and on the side of the pulmonary tissue by fibrous tissue of new formation. The study of sections cut transversely across the equator of ampullar dilatations, or progressively from the parts less dilated to the narrow parts which separate the ampullæ, confirms the description given. In the first no trace of smooth muscle cells can be found; in the latter rather large muscular fasciculi are found, the fibres of which are cut across. The cause and evolution of bronchial dilatation can now be easily understood. In consequence of acute and long continued bronchitis, which has affected the wall of a bronchus throughout its whole thickness, the elastic fibres and muscle cells become partly destroyed, and the bronchus undergoes dilatation.

In the foregoing histological description we had more particularly in view the dilatation of bronchi more than 1 mm. in diameter, and we have still to describe the changes which take place in dilatations which occur in the course of still smaller bronchi. These dilatations, ampullar in form, are often situated on the surface of the lung beneath the pleura. By their internal surface being bathed in pus or muco-pus, they are sometimes difficult to recognise with the naked eye. They communicate, it is true, with a bronchus, but they greatly resemble tubercular vomicæ. A bronchial dilatation is suspected if the surface is smooth and regular, or if glistening elastic fibres can be found, but it is made certain if, on microscopical examination of sections cut perpendicularly to the surface of the cavity, a more or less regular lining of cylindrical cells is found, and a basement membrane. The wall of such a sac only shows embryonic connective tissue if the lesion be rather recent, and lamellar if it be of old standing. Dilatations of the lobular bronchi are consecutive to capillary bronchitis, probably accompanied with foci of

lobular pneumonia, if the primary lesion has been purely inflammatory or of a tubercular character. It is much more probable that these dilatations are not only consequent to bronchitis, but are due to suppurative peribronchitis which has destroyed an entire lobule and terminated by interstitial pneumonia.

When the mucous membrane of a dilated bronchus is the seat of intense and puriform catarrh, it becomes red and vascular, loses its polish, becomes thick and covered with a large number of small papillary growths. When the formation of pus cells is very active the epithelium falls off, and a more or less deep and extensive **ulceration** is produced. The cavity contains a thick, grumous muco-pus; the mucous membrane is destroyed, and no trace can be seen of the primitive structure of the bronchus. Sometimes there is a greyish layer adhering to the internal surface of the cavity, formed of connective tissue in a state of mortification and infiltrated with more or less granular pus cells. This is the **superficial gangrene**, which gives, during the life of the patient, an extremely fœtid odour to the breath; this condition is caused by excessive purulent infiltration compressing the blood-vessels. The blood then coagulates, and the tissues, deprived of nutrition, die and become decomposed in the presence of the air in the cavity, and putrid products are formed. The pus is rather fluid, and of a yellowish-grey colour; the pus cells are filled with fat granules, beside which are often found crystals of margarin, cholesterin, leucin, and tyrosin. This pus is easily miscible with water, thus indicating that it contains but little mucin. On standing it separates into three layers. The upper is frothy and aerated, the middle clear, and the inferior is opaque and contains solid elements—that is to say, the pus cells and crystals mentioned above. The sputa show the same characteristics.

With cavities hollowed in the lung, it has often to be decided whether they are bronchial dilatations or tubercular vomicæ. On this question we may remark that dilated bronchi are often observed at the same time that vomicæ are being formed; further, that in the indurated pulmonary tissue which forms the wall of perfectly characteristic bronchial dilatations, old tubercular granulations are often found, which are fibrous, or formed of a fibrous tissue infiltrated with black pigment, in the midst of which are found giant cells mixed with melanic granules. On finding bronchial dilatations in a post-mortem examination it should always be ascertained what is the degree of evolution of the pulmonary tubercles. It is known that tuberculosis is a frequent

cause of bronchial dilatations, but it has not yet been determined to what degree they contribute to their development.

The lesions proper to bronchial dilatations, or which are caused by them, are slow in progress; the wall of a dilated bronchus may even undergo calcification (Schräder). More frequently the dilated bronchi, isolated from the rest of the bronchial tubes in the indurated apex of a lung, for example, are filled with a yellowish, almost solid mass, composed of caseous pus. After removing the contents the more or less modified structure of the bronchial wall can be recognised.

Ulceration of the bronchi.—Superficial ulceration of the bronchi is observed in the acute bronchitis which accompanies metastatic abscesses of the lung, purulent infection, puerperal fever, typhoid fever, glanders, &c.; it may also be consequent to variolic pustules, but its most frequent causes are tuberculosis and pulmonary gangrene. Abscess, gangrene, and tubercle of the lung may cause perforation of the bronchi; it is the same with bronchial ulcerations, whatever may be their form. It may be added that aneurism of the aorta, tumours (carcinoma, epithelioma, sarcoma), pleurisy, and suppuration of the bronchial glands affected with caseous degeneration are also causes of perforation.

Tumours of the bronchi.—A **lipoma** has once been observed by Rokitansky, developed in the submucous cellular tissue of the left bronchus and projecting into its cavity.

Calcareous incrustation and true ossification of the bronchial cartilages is not extremely rare in old persons who have had chronic bronchitis; it is found at the level of the division of the trachea and in the first bronchi. These tubes then become absolutely rigid. A similar process may occur in the small bronchi; but all the osseous needles, even those which are hollow, which are found accidentally in the lungs, particularly in the indurated apices in interstitial pneumonia, must not be referred to ossification of the bronchi or of their cartilages. As we have already explained (*vide* vol. i. p. 229), and as we shall see when treating of ossification of the lung, these growths are developed in the connective or embryonic tissue which characterises interstitial pneumonia.

Carcinoma is not developed primarily in the bronchi, but it may appear by extension of a tumour of the mediastinum, the lung, the œsophagus, or the bronchial glands. We have also frequently seen small, semitransparent, hemispherical tumours,

varying in size from a millet-seed to a lentil, projecting on the surface of the bronchial mucous membrane, and which resembled carcinoma in structure; they were secondary growths, resulting from scirrhus of the breast.

Tubercle.—Tubercle of the bronchi, which is so common and of such great importance in the study of pulmonary tuberculosis, should be examined in bronchi of different calibre, for the lesions which they cause differ according to the diameter of the tubes. In the large bronchi the tubercles have the same arrangement as in the trachea (*vide* p. 61). The tubercular granulations of the mucous membrane look like small whitish spots, which on ulcerating form a crater-like depression; they are isolated or confluent, and they are succeeded by serpiginous ulcerations. The mucous membrane is infiltrated for a varying depth, and the cartilages may be even uncovered by ulceration and become inflamed and ossified; in the bronchial cavity it is red and decidedly thickened. On microscopic examination it shows the lesions of the most acute inflammation. All the histological details already given of tuberculosis of the trachea and larynx are observed in tuberculosis of the large bronchi.

In the bronchi of medium calibre the tubercular granulations are rarely so evident to the naked eye; tubercles and tubercular ulcerations can, however, be formed. These lesions are accompanied with inflammatory troubles; the epithelial investment is modified, as it is in all acute inflammation (*vide* p. 62). The submucous tissue and the layer of smooth muscular fibres are infiltrated with lymph cells. Hence it is that bronchial dilatations are often found in tuberculosis, particularly at the moment when cavities are formed as the result of tubercular broncho-pneumonia. Often also in bronchitis related to tuberculosis irregular growths varying in size, and composed of embryonic tissue covered with epithelial cells more or less normal in type, are observed either at the level of the ulcerations or in the inflamed but not ulcerated submucous tissue. The bronchi are destroyed and divided at the point which corresponds to a cavity when the latter occupies the place of a lobule or many lobules in which the bronchi terminate. If the cavity is situated, not at the terminal extremity of a bronchus, but on one of its divisions, the loss of substance by means of which the cavity communicates with the bronchus is situated laterally.

The lesions of the lobular bronchi, which are less than 1 mm. in diameter, which possess neither glands nor cartilages, and have

only a very delicate fibro-muscular coat, blend with those of the lung in tubercular broncho-pneumonia. In sections of lung, made through foci affected with recent broncho-pneumonia, the small bronchi seem filled with lymph cells mixed more or less with epithelial cells. The muco-pus which they contain is derived partly from the mucous membrane lining them and partly from the acinous bronchus and the alveoli. The epithelial investment of the diseased bronchi is generally preserved, either wholly or in part. The connective tissue of the bronchial wall is infiltrated with lymph cells, so that its normal thickness is much increased and its calibre diminished. The blood-vessels of the bronchus are dilated and filled with blood. If the section passes through a tubercle the wall of the small bronchus shows at one or more

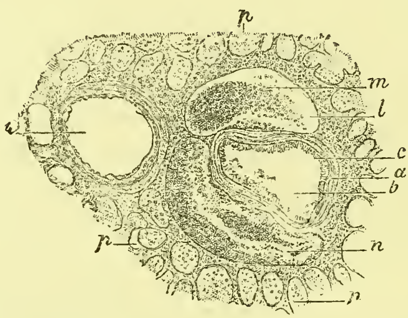


Fig. 36.—Inflammation of the Peribronchial Lymph Vessels, from a case of Tubercular Broncho-pneumonia.

a wall, *b* cavity, and *c* epithelium of a bronchus; *l*, *n*, peribronchial lymph vessels, the dilated cavity of which is filled with cells, *m*; *v*, arteriole; *p*, *p*, alveoli of the lung. Magnified 20 diameters.

points of its circumference an islet of small cells, in the midst of which the capillaries are obliterated, as in every tubercular granulation. These peribronchial tubercles are especially found, according to Rindfleisch and Charcot, at the level of the spur which the bronchi form when they divide into lobular bronchi. The wall of the arteriole which accompanies the small bronchus is inflamed and exhibits both endo-arteritis and peri-arteritis. The peribronchial lymph vessels, the volume of which may be considerable, are equally inflamed (*vide* fig. 36) and filled with lymph cells. These lesions almost always accompany tuberculisation of the bronchial glands.

Tubercular bronchitis is intimately related to tuberculosis of the lung itself; the lobule in which the bronchus ends is the seat of tubercular granulations and of pneumonia, with or without

fibrinous exudation into its alveoli. Later, when the tubercular granulations become caseous, when their capillary vessels are com-

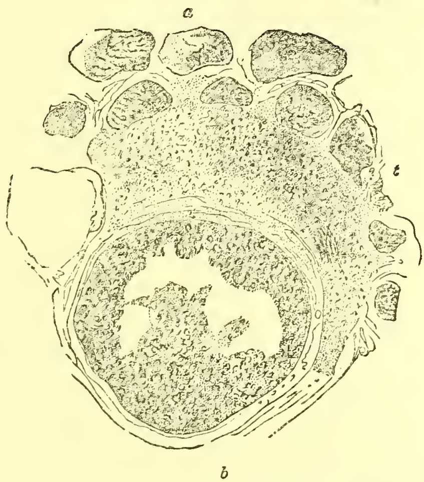


Fig. 37.—Section of a Focus of Tubercular Broncho-pneumonia.

The bronchus, *b*, is filled with caseous pus; it is surrounded at its upper part by a tubercular granulation and by alveoli, *a*, filled with pneumonic exudation. Magnified 40 diameters.

pletely obliterated, and the course of the blood in the arterioles has been arrested either by the obstruction the circulation meets

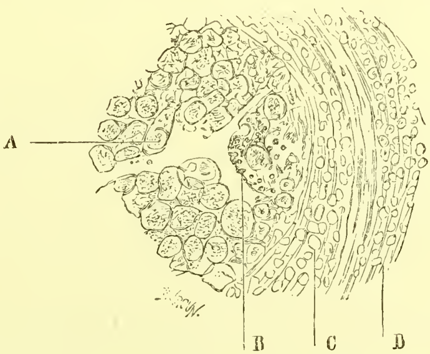


Fig. 38.—Section of a Bronchus affected with Tubercular Bronchitis.

A, tumefied, round, and fatty cells contained in the bronchus; B, cells in a state of fatty degeneration; C, submucous connective tissue; and D, peribronchial connective tissue, containing a large quantity of small round, embryonic cells. Magnified 150 diameters.

with in the capillaries or by endo-arteritis, all the tissue comprised in a pulmonary lobule is doomed to destruction. The epithelial and lymph cells contained in the small bronchus blend

together, become dry, and filled with fine granules, and finally undergo a kind of molecular disintegration. The wall of the bronchus, caseous itself, undergoes the same fate and ulcerates at the same time as the entire lobule. This is one of the most usual modes by which small tubercular cavities in the lung are formed.

V. Lesions of the Lung.

Anæmia.—Pulmonary anæmia is present in cholera and in general cachectic diseases. At other times it is caused by compression or retraction of a more or less considerable part of the lung, in consequence of a pleuritic effusion for example; the lung is then extremely pale, its vessels are empty of blood, but it does not show any other marked lesion. The lung is generally considered to be atrophied if it has undergone a considerable shrinking when pressed upon by an abundant pleuritic effusion and maintained in its new position by thick false membranes. But we have not here a true atrophy, for though the organ is diminished in size its essential parts are preserved. The air being expelled from the vesicles, the alveolar walls are simply squeezed close together. In pulmonary emphysema there is, on the contrary, much more actual atrophy, for the septa of the alveoli have disappeared, to a certain extent, by a process of atrophy; this disease, however, causes an increase in size of the diseased organ.

Atalectasia. Pulmonary collapse, fœtal condition.—Atalectasia, that state of the lung in which the pulmonary alveoli are empty of air, is caused principally by two diseases; first, bronchitis, which prevents the air entering the bronchi; secondly, thoracic effusions, in which the pleural fluid compresses the lung. The extreme degree of this condition is observed in considerable pleuritic effusions.

Atalectasia caused by a lesion of the bronchi is recognised by areas of slight depression, most frequently found on the surface of the lung. These depressed spots are soft, fleshy, of elastic resistance, and of a violet-red colour. On cutting out a piece of such a lung and placing it in water it sinks to the bottom, and it does not crepitate when rubbed between the fingers. On dividing the lung the pulmonary tissue appears of a dark red colour, smooth and uniform in texture, and only a small quantity of fluid can be squeezed out on pressure. The atalectasic parts greatly resemble

the lung of a fœtus which has not yet breathed, whence the name, the fœtal condition, which has been given it by the French authors (Legendre and Bailly, Rillet and Barthez, &c.) After insufflation the alveoli return to their normal condition. This form of atalectasia is observed in simple or capillary bronchitis, lobular pneumonia, typhoid fever, phthisis, &c. It is situated particularly on the periphery of the lung at its anterior and posterior borders, and in its middle or inferior lobes; the pleural adhesions which are so frequently present at the level of the superior lobe prevent collapse of this part of the lung.

When atalectasia is due to pressure caused by a pleuritic effusion, a considerable part of the inferior lobe, or possibly the whole lobe, or even a whole lung, will be found flattened or globular, empty of air, anæmic, and giving no longer a sensation of crepitation between the fingers. At the level of the parts compressed by the effusion, the pleura will be found to be almost always thickened, opaque, and of fibrous consistence, and fibrinous or organised false membranes will often be present. When the lung is thus surrounded by a fibrous shell formed by the thickened pleura and false membranes, it is impossible to restore it to its normal condition by insufflation; but if a thin slice of this lung be taken and extended on a glass slide the pulmonary parenchyma will be seen to be preserved, and the alveoli will return to their primitive condition. After hardening atalectasic tissue in alcohol or in Müller's fluid, gum and alcohol, and on examining sections under the microscope, the alveoli are seen to be flattened; the capillaries which form the network on the alveolar wall project and are filled with blood; the epithelial cells of the alveoli are globular, their nuclei large, and their protoplasm granular. The alveolar cavity often contains a small quantity of fluid, round, granular, or pigmented cells, lymph cells, and a few red blood corpuscles.

When pulmonary collapse is of bronchial origin it is caused by the obstruction of a small bronchus by a plug of mucus. This collapse is explained in the following manner: A plug of mucus is present in a small bronchus; by an effort of respiration it is drawn into a still smaller bronchus, where it is arrested (for example, at the spot where the bronchus divides); air cannot pass it in inspiration, but during expiration the air contained in the infundibulum below the mucous plug pushes it into a larger tube and is thus able to pass. At the next inspiration the plug of mucus descends again, and prevents the air from penetrating into the

infundibula which are below it. At the next expiration the plug of mucus again allows the air contained in the infundibula below it to pass, and so on till the alveoli are gradually emptied. It is also probable that part of the air which they contain is absorbed. Finally, the pulmonary vesicles contain no air, and a complete atalectasia of the lobule which corresponds to the obstructed bronchus ensues.

The mechanism of atalectasia by peripheral pressure due to pleuritic effusion is different. The lung cannot dilate, for it is impossible for it to push back the abundant fluid which fills the pleura. The pressure exercised by the fluid causes the lung to become gradually emptied during expiration of the residual air which the pulmonary alveoli contain. Finally, the thickening of the visceral pleura, which is caused by the pleurisy, changes this membrane into a fibrous sac, which keeps the lung in a more or less completely collapsed condition. In this condition longitudinal folds are seen on the surface of the pleura and of the subjacent lung. The entire lung is reduced to a thin, flattened, flabby, and elastic strip, which sinks to the bottom in water. Often the pleurisy which has caused this complete atalectasia is itself due to a special inflammation or to new growths in the lung, as, for example, tubercle, a pulmonary cavity opening into the pleura, pulmonary gangrene ending in pyo-pneumothorax, to septicæmic infarctus following typhoid fever, and pulmonary apoplectic foci, as may be seen in cardiac diseases, &c.

In these various conditions the collapsed lung shows different lesions; sometimes vomicæ, confluent tubercle in a more or less caseous condition, sometimes gangrenous cavities, or purulent infarctus, or apoplectic foci. The pulmonary tissue is generally pale and anæmic, as is observed in most effusions due to pleurisy or in those complicating tuberculosis; it is very red and congested when due to an effusion which complicates pulmonary apoplexy, but in every case it is always completely deprived of air.

Hyperæmia of the lung.—Hyperæmia of the lung is an extremely frequent lesion; it is seen, in fact, in most acute febrile diseases, such as typhoid fever, measles, scarlatina, erysipelas, &c., and in all diseases of the heart, lungs, and bronchi (acute bronchitis, broncho-pneumonia, pneumonia, tuberculosis, emphysema, &c.) It occurs, moreover, a short time before the death agony, or during the agony itself, at the end of all diseases, and in cases of violent death which are accompanied with a more or less marked

degree of asphyxia. The congested lung is shrunken, smooth and red on the surface; on section it is also seen to be red, and in all cases of congestion caused by asphyxia, more or less extensive and numerous ecchymoses are often found on the surface of the pleura. The pulmonary tissue is infiltrated with a red or pink frothy fluid, which may be squeezed from the bronchi and alveoli after cutting into the tissue. The bronchi are also generally very much congested and inflamed, and lined by a mucous or muco-purulent secretion which is often blood-stained.

On examining sections of a congested lung hardened in picric acid or alcohol, the capillary vessels are seen to be filled with blood coagulated by the hardening reagents, and they form sinuous loops and bends along the wall of the alveoli, and project into the interior of these cavities. In consequence of active or passive congestion the epithelial cells lining the blood-vessels and the internal surface of the alveoli become tumefied, swollen, and granular, and undergo a series of nutritive modifications. These turgid, granular, or vesicular cells, measuring from $15\,\mu$ to $30\,\mu$, are often of a yellowish colour, due to absorption of the hæmoglobin dissolved in the fluid which fills the pulmonary alveoli. Most of these cells generally fall into the alveoli, where they become free and spherical; they may even sometimes contain yellow or red pigment granules, derived from the blood, or fine black granules. The fluid infused into the interior of the alveoli contains at the same time a few red blood corpuscles and also white corpuscles. The protoplasm of the latter sometimes devours the red blood corpuscles, which are seen in them, at times entire and perfectly recognisable and at others broken up. The epithelial cells which are still attached to the alveolar wall, as well as those which have become free, often contain two or more nuclei. In less acute congestion, of whatever cause, a very fine reticulum of fibrin is often found in sections cut from hardened specimens composed of fibrils between which are found red and white blood corpuscles and epithelial cells. This presence of fibrin is due to extravasation of the plasma from the blood-vessels, from which it escapes at the same time as the formed elements of the blood. Fibrin and red blood corpuscles are found in the pulmonary alveoli in the so-called hypostatic congestion due to chronic cardiac disease, as well as in the congestion due to an acute pulmonary affection which is regarded as active.

Between congestion and the commencement of true inflammation and pneumonia it is impossible to distinguish. All

pneumonia is, in fact, preceded by pulmonary congestion, that is to say, by a period in which the capillary vessels of the alveoli are distended with blood, and diapedesis of the elements of the blood into the alveoli occurs at the same time that the epithelial cells are modified, undergo proliferation, and desquamate. The lesion is the same at the commencement of catarrhal pneumonia and fibrinous pneumonia as in simple pulmonary congestion, slight as this may be. The congestion may often cause, as we have already shown, the formation of fibrinous coagulation in the midst of the liquid exudation which fills the alveoli (fig. 39). There is then in reality a series of anatomical conditions which

FIG. 39.—CONTENTS AND WALL OF A PULMONARY ALVEOLUS IN CONGESTION OF THE LUNG.

a, fibrils of fibrin enclosing in the network they form red blood corpuscles, *c*, and lymph cells, *b*; *d*, an epithelial cell detached from the wall; *g*, capillary vessels of the alveolar wall containing red blood corpuscles; *e*, lymph cells attached to the wall; *h*, an epithelial cell near the wall. Magnified 300 diameters.

establish an insensible transition from congestion, marked only by repletion of the blood-vessels, to acute congestion, when the alveoli are filled by a fluid which contains a few epithelial cells and elements of the blood in the midst of a network of delicate fibrils of fibrin: this latter condition constitutes pneumonia. In our opinion pneumonia is not established till the pulmonary alveoli no longer contain air, but are completely filled with lymph and epithelial cells, or with an exudation formed of cells and fibrin. Active congestion cannot be microscopically distinguished from passive or hypostatic congestion; the lesions are the same in the

two cases. The pulmonary congestion of typhoid fever, which has, wrongly we think, been regarded as passive, generally shows all the histological characters of most acute congestion, for a rather large quantity of lymph cells and a reticulum of fibrin are generally found in the alveoli; further, this congestion always accompanies the bronchitis, in which the connective tissue of the bronchial mucous membrane is infiltrated with lymph cells; and, finally, side by side with congestion catarrhal pneumonia is often found, and even lobar fibrinous pneumonia.

Chronic hyperæmia—black pigmentation of the lung.—Chronic hyperæmia, or rather repeated and successive congestion of the lung, causes more marked and permanent modifications. We have

FIG. 40.—SECTION OF A LUNG AFFECTED WITH INTERSTITIAL PNEUMONIA, WITH PIGMENTATION OF THE CELLS.

a, pigmented lymph cells situated in an alveolar septum; *b*, *b*, *b*, tumefied epithelial cells forming a delicate membrane on the surface of the alveolar wall, from which it is partly detached; *n*, *n*, *n*, pigmented round cells in the centre of the alveolar cavity. Magnified 300 diameters.

already seen how the epithelial cells become pigmented; the same pigmentation may be produced in the alveolar walls; the capillary vessels exude into the substance of the alveolar walls a fluid containing red blood corpuscles; the connective-tissue cells swell and pigment granules are deposited within or around them. It is easily

understood that the more frequent and persistent is this congestion the more marked is the pigmentation of the connective tissue of the lung; and it may be seen at its maximum in cardiac diseases accompanied with great impediment of the pulmonary circulation, particularly in regurgitation or stenosis of the mitral valve. When hyperæmia of the alveolar walls is very active or prolonged, the connective-tissue elements tend to proliferate and form a new fibrous tissue, principally around the bronchi and vessels. The first stage of interstitial pneumonia is characterised by this thickening of the pulmonary tissue. The black discolouration of the lung may be also due to another cause, namely, to penetration into the ultimate vesicles of the bronchi, and into the pulmonary parenchyma itself, of fine particles drawn in with the air; this may be observed in artisans who are constantly exposed to the action of different kinds of dust —colliers, miners, lapidaries, workers in stone, copper, steel, modellers, &c. The penetration of particles of carbon, doubted by Virchow, who formerly attributed all pulmonary pigmentation to changes in the colouring matter of blood, was demonstrated when Traube and Rindfleisch found, in the alveoli of a workman employed in charcoal-burning, fragments of vegetable cells, perfectly recognisable by their porous canals. The disease called anthracosis will be studied in detail when treating of interstitial pneumonia.

Œdema of the lung.—In œdema of the lung the surface of the organ is stretched; it is swollen, and on incising it or pressing it between the fingers a rather large quantity of a transparent, frothy and colourless fluid can be squeezed out. This condition is rather frequently met with in the course of the cachexiæ which cause anasarca, as, for example, in cancer and albuminuria. It is also developed by causes which favour passive congestion of the lung. In some cases the serum which runs out is pinkish; this is an intermediate state between congestion and œdema. The distinction between these two lesions is, however, of slight importance, since they may be produced under the same condition, that is to say, when the circulation is impeded (emphysema, cardiac diseases, tuberculosis, &c.) The seat of œdema is the same as that of passive congestion, that is to say, the inferior lobe and the posterior border of the lungs. To the dorsal decubitus after death is probably due the accumulation of fluid in the most dependent parts of the lungs. On examining sections of an

œdematous lung, fluid, bubbles of air, a few red blood corpuscles, lymph cells, and free epithelial cells are found in the alveoli; sometimes also very delicate filaments of fibrin arranged in a large meshed network may be observed.

Pulmonary apoplexy.—Pulmonary apoplexy consists essentially in extravasation of blood into the alveoli and small bronchi. This lesion is observed most frequently in cardiac diseases, particularly in those of the mitral valve; it is sometimes related to eruptive fevers—scurvy, typhoid fever, &c. When the blood pressure in the capillary system of the lung is much increased, particularly when the blood finds great difficulty in returning from the lungs to the heart in consequence of increased tension in the left auricle, passive congestion of the lung is produced, which ends in rupture of some of the capillaries and flooding of the alveoli with blood. The parts of the lung affected with apoplexy are tense, absolutely empty of air, do not crepitate, and if thrown into water immediately fall to the bottom. The foci are generally spherical or in the form of a cone, with the base directed towards the pleura; on section they are of a deep red, like venous blood, or of a sepia tint. On carefully observing this section it is seen to be granular. The opened bronchi contain a red, blood-stained mucus; the arterioles and small veins of the affected part are thrombosed, and filled with a clot, which is adherent to the vascular walls; this coagulum is red when recent and discoloured when old. The zone of lung which surrounds the apoplectic foci is red, highly congested, or affected with pneumonia.

After hardening a part of the lung thus altered in alcohol or chromic acid, and making delicate sections, it will be observed that the alveoli are filled with red blood corpuscles, which, flattened and deformed by reciprocal pressure, form a kind of mosaic of quite a typical appearance. Under the influence of the hardening fluid, particularly of alcohol, the appearance of the red blood corpuscle is, in fact, completely changed; instead of appearing, as in the normal condition, in the form of a flattened but full disc, it now resembles a small vesicle; and in consequence of the pressure these elements exercise on one another their outlines, instead of being circular, become polygonal; hence the appearance of the mosaic. In the midst of this mosaic large granular and spherical cells are always found, which may be isolated or grouped together. These cells almost always contain yellow or red pigment granules if the extravasation be recent, and black if the

apoplexy be old. Crystals of hæmatoidin may also be found. In preparations stained with carmine these cells are the only elements which take the colour; they are derived either from the epithelium of the alveolus or they are lymph cells which have absorbed neighbouring red corpuscles, and have become hypertrophied; they are disseminated throughout all the alveoli, either at their borders or in their centre, and they pass also into the bronchi, from which they are expelled with the blood-stained mucus that these tubes contain. In the sputa of pulmonary apoplexy the same granular elements may be met with floating in

FIG. 41.—SECTION OF A LUNG AFFECTED AT THE SAME TIME WITH INTERSTITIAL PNEUMONIA AND PULMONARY HÆMORRHAGE. Magnified 200 diameters.

The septa of the alveoli are thickened, and in the interior may be seen large round pigment cells, polyhedric cells, and red blood corpuscles.

a red, mucous, almost colloid fluid, which also contains a large number of red blood corpuscles. The final bronchial ramifications are completely filled with coagulated blood. In recent apoplexy a fine reticulum of fibrin, including red blood corpuscles and the large round cells contained in the alveoli and the ultimate bronchi, may also be found on microscopical examination; but at the end of a few days the trabeculæ of fibrin have disappeared. The surface of the lung on section is granular in appearance, which

is due to the complete plugging of the infundibula and the alveoli with coagulated blood. Each infundibulum forms with the alveoli, which lead into it, a system of cavities which, filled by the solid clot, looks like a small isolated granule, and forms a relief visible to the naked eye, measuring $\frac{1}{2}$ mm. in diameter. These grains or granules, caused by the blocking of the infundibula, placed one against the other, and separated only by the pulmonary septa, the small vessels, and bronchi, give a granular appearance to the surface of the lung on section. We may remark in passing that it is the blocking of the infundibula by fibrin and pus which constitutes the spots in pneumonia. In apoplexy the spots are of a deep red colour, stained by the venous blood, whilst they are pinkish or grey in fibrinous pneumonia. The blood vessels of the whole of the diseased part are filled with coagulated blood. On examining a section of an arteriole it may be seen that it is completely filled by concentric layers of fibrin (fig. 42); the most external adhering

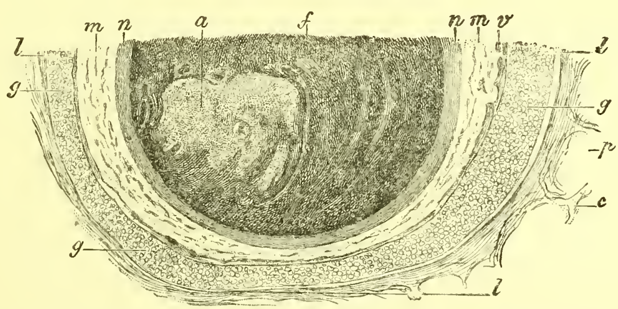


Fig. 42.—Section of a Vessel of the Lung surrounded with a Lymphatic Sheath in Pulmonary Apoplexy.

f, fibrinous and cruoric coagulum arranged in concentric layers; *a*, central portion of the clot; *n*, internal coat of the vessel; *m*, middle coat; *v*, vasa vasorum; *l*, *l*, lymphatic filled with red blood corpuscles, *g*; *p*, pulmonary alveolus; *c*, alveolar wall. Magnified 80 diameters.

to the internal coat are more or less exactly parallel, but those situated towards the centre of the vessel are often quite irregular. The internal wall of the arteriole is inflamed if the thrombosis has continued a certain time. A rather curious fact, which one of us has observed many times, is that, in pulmonary apoplexy, the lymph vessels of the lung are filled with coagulated blood and distended quite as much as the pulmonary alveoli; thus the lymph vessel *l*, which surrounds the blood vessel in fig. 42, is completely filled with red blood corpuscles. The facility with

which blood passes into the pulmonary lymphatics is due to the fact that they communicate with the alveoli. It is probable that the extravasation of blood into the pulmonary alveoli is due to the rupture of some of the capillaries, but neither rents nor stomata can be discovered under the microscope by means of which the red blood corpuscles could pass.

Pulmonary apoplexy may present the following different appearances to the naked eye.

First, **Hæmoptic infarctus of Laennec.**—This infarctus is characterised by one or more firm foci, of a brown or sepia tint, generally circumscribed so that there is a sharp line of demarcation between the indurated point and the healthy or congested tissue which surrounds it. On incising the altered tissue it is seen that the surface of the section is of a deep red tint, granular in aspect, and on pressing it a small quantity of thick fluid, containing no air-bubbles, is squeezed out with difficulty. The surrounding tissue is often supple, crepitating, or may be slightly infiltrated with blood. The bronchi leading to this part contain a thick blood-stained mucus, which contains, like the sputa, blood and spherical cells, the latter infiltrated with fat and pigment granules. The blood vessels visible to the naked eye are filled with clot, which is red if the apoplexy be recent, and fibrinous and discoloured if the lesion be older. The most frequent seat of these infarctuses is the centre of the inferior lobe, or near the root of the lung; they are also often superficial, occupying the sharp edge of the lung, when they may be seen through the pleura. When they are situated beneath the pleura, they make a slight projection, and at their level the serous membrane is inflamed and covered with fibrinous false membranes; there is then often also an effusion into the pleura of a sero-fibrinous fluid more or less mixed with blood; if this effusion be very abundant the lung may be compressed and collapsed, which condition would not be discovered except by an attentive examination of the apoplectic foci where it is seated.

Secondly, **Focal apoplexy** is caused by the sudden effusion of a large quantity of blood into the lung. It probably results from the rupture of one or more of the large blood vessels. In this case a mass composed of coagulum and fluid blood is found in the midst of a pulmonary lobe surrounded with flakes of torn pulmonary tissue; this is an actual apoplectic focus, similar to those produced in the brain. If the apoplexy is situated at the periphery of the lung, the pleura is torn, and blood flows into

the pleural cavity. This form of apoplexy is very rare and is rapidly fatal.

Emphysema of the lung.—Emphysema of the lung is characterised by the formation of cavities or vacuoles, varying in size, and which result from the dilatation or confluence of alveoli, the walls of which have totally or partially lost their elasticity. An emphysematous lung appears to the naked eye to be tumefied, tense, inflated, and with an irregular surface; at its apex or borders vesicles with very delicate walls are often seen projecting under the pleura, varying in size from that of a small pea to a nut, or even larger; they are full of air, and remain inflated after the thorax is opened, but on pricking them they partly collapse. The irregular surface of an emphysematous lung is pale and anæmic. On squeezing it between the fingers it gives the sensation of elastic resistance, and a certain softness, which has been compared to that of a down pillow. When the lungs are only partially affected by emphysema, it is generally seated in the superior lobe, the internal and lower borders of the lung, or anteriorly. The posterior part of the lung and its inferior lobe and posterior border are more rarely affected; most frequently in emphysematous subjects these latter regions are congested. Sections made through the emphysematous parts show vacuoles varying in size, generally visible to the naked eye, and which have taken the place of the normal infundibula. It was thought for a long time that, in pulmonary emphysema, the alveoli had simply become larger; but there is now no doubt that this alteration results from thinning of a certain number of the alveolar walls, the septa of which atrophy and disappear. On removing the wall of a large emphysematous vesicle with the scissors and simply spreading it on a slide, vestiges of the elastic septa of the primitive alveoli may be recognised opening into one another.

On examining under the microscope sections of an emphysematous lung which has been inflated and dried, one is struck with the considerable increase in diameter of the pulmonary infundibula. The septa of the alveoli which open into the infundibula project, on the contrary, less than in the normal condition, and it will be easily seen, both in sections of an inflated and dried lung, as well as in portions of the lung removed with scissors in the fresh condition, that the interalveolar septa are often perforated. In the first stage, emphysema simply affects the infundibulum. Its cavity is increased at the expense of the

alveoli which open into it, the more or less atrophied septa of these having disappeared. When an emphysematous infundibulum is seated under the pleura the absence of resistance enables it to dilate more; the largest of the vacuoles may attain the size of a walnut, owing to the communication of neighbouring infundibula with one another: this is the most advanced stage of the disease. In some emphysematous subjects, particularly in old persons, the pulmonary tissue is converted at the apex and anterior border of the superior lobes into lacunæ communicating with one another, so that on pressing any point of the organ air can be made to circulate in almost every direction. Perforation of the septa is produced in this way; they are, it is known, composed of a very delicate membrane which contains elastic fibres, and covered by a close network of capillaries, lined with an epithelium the nuclei of which occupy the spaces or dents which are limited by the capillary vessels. Villemin, though he mistook the signi-

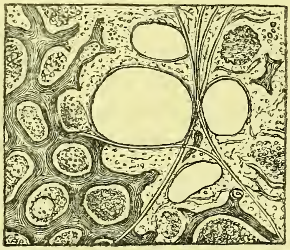


Fig. 43.—The Membrane of a Pulmonary Alveolus showing many Solutions of Continuity, from a Case of Emphysema, after Villemin.

fication of the epithelial cells, which he looked upon as connective-tissue cells, has, nevertheless, well described and appreciated this process in all its essential characters. As the result of a process still misunderstood, the nuclei situated between the capillaries hypertrophy, become granular, and soon, in the place of a living element, is found a small mass of organic matter killed by fatty degeneration. This mass, deprived of all vitality, is soon destroyed, and leaves in its place a perforation of the wall, which brings about a communication between two neighbouring alveoli which were hitherto independent. Many similar perforations are soon seen simultaneously in the same septum. The elastic fibres, which continually tend to retract, cause rents, and the capillary vessels, deprived of their support, undergo atrophy, and hence vascularisation is diminished or altogether abolished at these

points. An infundibulum, thus altered by the simultaneous lesion of many of the alveoli which open into it, soon shows, on its inner wall, instead of the septa which separated the alveoli, simple projections formed by the elastic fibres which are the last vestiges of these septa. Villemin concludes from these facts that emphysema consists solely in a lesion of nutrition, and that pressure of the air on the septa is not necessary for its production.

On removing with scissors the delicate wall of an emphysematous cavity projecting under the pleura, and on staining its internal surface with nitrate of silver, the flattened epithelium which lines the emphysematous dilatation may be observed. The epithelial cells, which are examined in situ in preparations which have not been stained with nitrate of silver, often show fatty granules in the protoplasm around their nucleus, as Rindfleisch has described. Thinned septa also show, in their interior and on their surface, ovoid masses of fat granules lodged in the epithelial cells, or sometimes in the cells of the capillaries. It is probable that this granular degeneration plays a great part in the formation of small perforations in the interalveolar septa.

Senile emphysema is certainly chiefly characterised by lesions of nutrition of the lung, but there is nothing to prove that repeated bronchitis, cardiac diseases, &c., may not be the initial cause of these lesions, for we find in croup, whooping cough, the broncho-pneumonia of children, tuberculosis, &c., indubitable causes of emphysema. In these diseases, in fact, the infundibula are dilated in consequence of the efforts of coughing, of inspiration and expiration; small vacuoles are thus produced, which are easily seen on the surface of the lung. These emphysematous vacuoles or vesicles are sometimes filled with mucus or mucopus, if the acute stage of bronchitis continues. Thus we are not able to allow, with Villemin, that emphysema is always an idiopathic disease primarily caused by a fault of nutrition.

In the walls of large emphysematous dilatations, and in the greater number of emphysematous lungs in old persons, a rather marked pigmentation may be observed in the walls of the vessels. In these cases we have searched in vain for the atheromatous changes in the vascular walls which have been hitherto rather more supposed than proved by many authors, in order to explain the production of idiopathic emphysema. On examining the internal surface of the wall of the emphysematous vacuoles projections are seen, formed of bundles of elastic fibres which belonged to the effaced alveoli, and which are pressed against the dilated

surface. If the emphysema extends over a great part of the lung or one pulmonary lobe, the diseased part is anæmic, owing to diminution of the circulation, while in the parts which remain healthy the tissue is red, œdematous, and gorged with blood. Emphysema is seated usually, as we have already said, in the superior lobes, and more particularly at their internal and inferior borders; and here it sometimes shows itself by bullæ filled with air, or even by globular appendices. When a lung is entirely emphysematous it is hypertrophied in a marked manner; it fills the pleural cavity, and on opening the thorax it does not shrink; sometimes even it may depress the liver and displace the heart. Emphysema may become the cause of pneumo-thorax by rupture of a vesicular dilatation; interlobular emphysema, due to penetration of air into the subpleural cellular tissue in consequence of rupture of the alveoli, is sometimes propagated to the mediastinum, and to the neck and subcutaneous cellular tissue, when it gives the visceral pleura the appearance of being raised up by froth. The bubbles of air forming the froth are easily displaced by pressure, and run under the pleura; this fact differentiates subpleural emphysema from that situated in the pulmonary vesicles.

The most frequent causes of emphysema are asthma, whooping cough, and generally all diseases of the chest accompanied with cough and violent efforts of inspiration and expiration. The question has been much discussed if emphysema is caused by the efforts of inspiration or expiration. Laennec thought that it was produced by inspiration. Jenner, Mendelssohn, and Jaccoud maintain, on the contrary, that it is caused by expiration, particularly when this is accompanied with effort, as, for example, in fits of coughing, when the glottis is partly closed. The recent experiments of Hirtz [1] are in favour of inspiration being the cause. Hirtz placed a ligature on the trachea of a rabbit, so as to reduce its calibre; the animal died emphysematous at the end of a week. Inspiration was seen to be particularly laboured to enable air to enter the lungs, but the obstacle was as great to expiration as to inspiration. To ascertain the part played in inspiration, Hirtz cut the phrenic nerves in the neck; the diaphragm no longer contracted, the force of inspiration was greatly diminished, and emphysema was not produced. Pulmonary emphysema is frequent in tubercular subjects, particularly in the case of fibrous tuberculosis accompanied with interstitial pneumonia.

[1] *De l'Emphysème chez les Tuberculeux.* Thèse de doctorat. Paris, 1878.

Senile atrophy of the lung, which is related, as we know, to interstitial pneumonia of the apex, is almost always accompanied with emphysema. J. Renaut and Bard[1] attribute emphysema to the cicatricial contraction which fibrous tubercles and islets of interstitial pneumonia produce in the walls of neighbouring alveoli, which thus become dilated.

Inflammation of the lung.—The forms of pneumonia or inflammation of the lung are numerous. Two species of pneumonia are distinguished, according as the changes caused by inflammation affect the contents of the alveoli or their walls. We shall describe in the first place lobular, lobar, and metastatic pneumonia, which are essentially characterised by an intra-alveolar exudation; then interstitial pneumonia, in which the lesion consists particularly in a change of the fibro-vascular framework of the lung. We shall study tubercular pneumonia with tubercle.

1. Broncho-pneumonia (lobular or catarrhal pneumonia, disseminated pneumonia, suffocating catarrh, capillary bronchitis, &c.) —Broncho-pneumonia, that is to say that collection of lesions of the lung which follow acute inflammation of the lobar or lobular bronchi, has received very different names, owing to its variety of manifestations. The anatomical lesions of broncho-pneumonia are numerous, and vary as regards extent and dissemination. They are not the same at different ages, nor in the various diseases of which broncho-pneumonia is an epiphenomenon. Broncho-pneumonia is much more frequent in children than in adults; its course and its evolution are subject to great variation; it is sometimes very acute, ending in a few days by the symptoms of suffocating catarrh and death; sometimes it is marked by successive irregular inflammatory abscesses invading both lungs; sometimes it is subacute or chronic, lasting many months or even years. Broncho-pneumonia may follow primary bronchitis, influenza, and eruptive fevers, measles, variola, diphtheria, erysipelas, typhoid fever, &c., and it often complicates pulmonary phthisis, as we shall see later on. Its different lesions are generalised bronchitis, bronchial dilatations, lobular bronchitis, nuclei of lobular pneumonia, peribronchial abscesses, yellow spots, vacuoles, hepatisation, catarrhal or epithelial pneumonia, fibrinous lobular or pseudo-lobar pneumonia, pulmonary congestion, atalectasia, emphysema and pleurisy. Among these lesions, which exist in a variable degree, and which

[1] *De la Phthisie fibreuse chronique.* Paris: J. B. Baillière, 1879.

are far from being always united in the same lung, there are some which we have already described, such as pulmonary congestion, atalectasia, dilatation of the bronchi and emphysema; we shall therefore now proceed to describe the others, and after studying these lesions singly we shall indicate their grouping, and which are the most usual anatomical and clinical forms of the disease studied as a whole.

A. **Lobular bronchitis and nuclei of lobular pneumonia.**—We have already described the essential lesions of acute lobular bronchitis, p. 62. We have seen how the divisions and subdivisions of the lobular bronchus and the terminal bronchi are filled and distended by lymph cells; that the epithelial lining is partly disintegrated, at the same time that the entire bronchial wall is infiltrated with lymph cells; the muscular elements of the wall are dissociated by pus, and are often irrevocably destroyed. The infundibula and alveoli are filled with lymph cells and with desquamated epithelial cells, which are granular, round, or cubical, and often multi-nucleated. Along the whole length of the principal bronchus of the lobule and its divisions, and at the extremity of the latter, the alveoli which are in contact with the bronchial tubes and which are continuous with them are full of lymph cells and epithelial cells. Inasmuch as inflammation is propagated by the central bronchus of the lobule, it is around it that inflammation begins in the alveoli, and where it is always the most acute; soon, in fact, the alveoli which surround a lobular bronchus and its divisions show in their interior a fibrinous exudation in the form of fine fibrils including lymph cells. Around the zone of peribronchial fibrinous pneumonia another zone may be observed, in which the alveoli contain lymph cells, red blood corpuscles, and tumefied epithelial cells which are adherent to their wall or free in their cavity. The inflammation progresses thus till the limit of the lobule is reached.

On hardening nuclei of commencing lobular pneumonia in Müller's fluid, gum and alcohol, and examining delicate sections under a low power, one or more opaque zones will be seen in the spaces which correspond to a lobule, in which the pulmonary alveoli are found to be completely filled with a fibrinous exudation; at the centre of each one of these is seen a section of a bronchus and the pulmonary artery which accompanies it; these are the peribronchial nodules of Charcot. If the section be cut perpendicularly to the bronchus, the section of the bronchus will be seen at the centre of the nodule, and it may be recognised by its

internal layer of cylindrical cells; its lumen is filled with pus cells; the fibro-muscular wall of the bronchus and its cellular sheath are infiltrated with lymph cells and changed into a large circular band in which it is impossible to recognise the normal elements; lymph cells only are seen separated by a few fibrils of connective tissue and by capillaries filled with blood. At a point in this peribronchial space the pulmonary artery may be found. The alveoli near the bronchus are filled with a fibrinous exudation, and around this zone of fibrinous pneumonia the alveoli simply contain

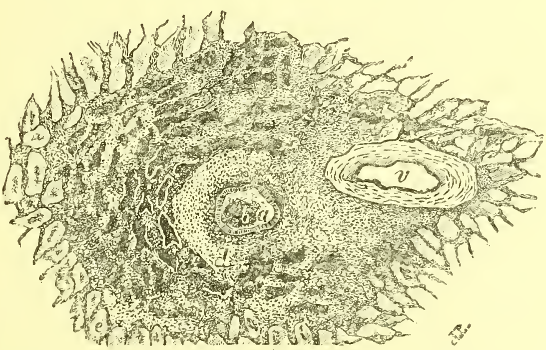


Fig. 44.—Section of a Nodule of Lobular Pneumonia, after Balzer.

b, section of a bronchus filled with lymph cells; *a*, its epithelial lining, formed of cylindrical cells; *v*, section of a branch of the pulmonary artery; *d*, peribronchial space filled with lymph cells; *m*, zone formed of alveoli filled with a fibrinous exudation; *e*, peripheral zone in which the alveoli are affected with catarrhal pneumonia. Magnified 20 diameters.

lymph and epitheloid cells (see fig. 44). In a more advanced period of inflammation, the band of connective tissue which surrounds the lumen of the bronchus, and which is infiltrated with pus, becomes thicker and more crowded with cells; the capillaries contain only lymph cells, and suppuration completely destroys the wall of the bronchus. Sometimes the alveoli which are contiguous to a bronchus are filled with pus cells, and the fibrin they primarily contain becomes granular. The septa of these alveoli soften and are destroyed, and thus along the terminal bronchi and the intralobular bronchus a peribronchial abscess is formed; we shall return later to this lesion. Thus in an inflamed lobule we find many peribronchial nodules showing the appearance, so well described by Balzer, of successive zones of fibrinous pneumonia, catarrhal pneumonia, and congestion. The alveoli affected with fibrinous pneumonia are squeezed and flattened by the pressure of the

thickened wall of the bronchus. The whole lobule is affected with catarrhal inflammation or hepatisation. Sometimes also the perilobular connective tissue is thickened, and shows a certain number of lymph cells. If the lobules affected by lobular pneumonia are seated at the periphery of the lung immediately under the pleura, and if a section be made of them perpendicular to the surface of the serous membrane, it will easily be seen that the connective tissue which limits the lobule on the side of the pleura is inflamed; further, that the subserous lymph vessels are always filled, at the level of the diseased lobules, with a large number of lymph cells, and sometimes with fibrin coagulated in the form of intersecting fibrils; finally, the pleura, which lines the superficial nodules of pneumonia, always shows the anatomical signs of pleurisy, characterised by the exudation of coagulated fibrin, which forms a network the meshes of which contain lymph cells.

These nodules of lobular pneumonia are recognised by the naked eye by thickening and induration of their tissue. On separating them entirely from the neighbouring parts, and putting them in water, they either sink to the bottom or float midway. In colour they vary from a deep red at the commencement of inflammation to grey or slightly yellowish when the inflammation is more advanced. When seated at the periphery of the lung they form marked projections under the pleura. They may be slightly dilated by inflammation, without the tissue taking, however, the normal appearance of lung; on crushing them the central bronchus is seen to be dilated and full of a yellowish and creamy pus; on section their surface is generally smooth, sometimes granular in places, of a red, grey, or yellowish colour, and a little blood-stained fluid or pus escapes. These nodules are generally of the size of a pulmonary lobule; they may, however, attain the size of a cob-nut, or even more by the union of many lobules. They are surrounded by pulmonary tissue which is sometimes very much congested and red on section, or sometimes simply yellowish.

B. **Peribronchial abscesses, yellow spots, vacuoles.**—Infiltration of the wall of the intralobular bronchi by a large number of lymph cells leads directly to the formation of peribronchial abscesses; for an abscess to be produced it is simply sufficient that the lesion should extend and that suppuration should reach the neighbouring alveoli, after destroying the wall of the bronchus. In a young woman who died from measles, we recently found in the middle of nodules of pneumonia yellow granules

which had the ramified and tubular form of the bronchi. In microscopic sections, coagulated pus was found in the centre of the abscess (*vide* fig. 45); the bronchial wall, in which not a vestige of cylindrical cells was found, was thickened and formed almost entirely of lymph cells; with a very high power, however,

FIG. 45.—SECTION OF A SUPPURATING BRONCHUS SURROUNDED BY PULMONARY TISSUE, ALSO SUPPURATING.

c, pus contained in the bronchus; *a*, *d*, wall of the bronchus infiltrated with pus (peribronchial space). The pulmonary tissue which surrounds the bronchus, *p*, *m*, is in a state of active suppuration. *m*, contents and *n*, wall of the alveoli, which contain a fibrinous exudation; *o*, intra-alveolar fibrinous exudation. The alveoli near the bronchus are flattened by pressure. Magnified 25 diameters.

a few fibrils of connective and elastic tissue were discovered. It recalled by its arrangement the band infiltrated with pus which surrounds an inflamed bronchus in a peribronchial nodule (*vide* fig. 44); at the periphery, the pulmonary alveoli were seen full of lymph cells, having partly lost their septa, and were blended with a mass of pus which had taken the place of the bronchial wall. At the periphery of these peribronchial abscesses, there was a zone formed of pulmonary alveoli filled with a fibrinous exudation; these alveoli were very obviously compressed and flattened by the abscess; finally, around this zone of fibrinous pneumonia catarrhal pneumonia was present. In other parts of these peribronchial abscesses not the slightest vestige of the primitive wall of the bronchi could be recognised, and the cavity of the abscess was circumscribed by alveoli affected with suppurative pneumonia. At the periphery also of these abscesses red blood corpuscles could not be seen in the capillary vessels, which latter were in a state of degeneration, and only contained a few white corpuscles.

These peribronchial abscesses (the mechanism of which has been so well described by Damaschino, Balzer, and Joffroy) cause pleurisy when they reach the surface of the pleura, or sometimes even perforation of this membrane. Yellow spots or purulent granulations may, as we have already said, result from peribronchial suppuration, or suppuration of many infundibula communicating with an inflamed terminal bronchus. On pricking one of these small granulations with the point of the scalpel, a drop of homogeneous pus escapes, which fact differentiates them from tubercle, with which they might possibly be confounded. Vacuoles, a very rare lesion by the way, are limited cavities situated on the surface of the lung or in its substance, and they communicate with the bronchi with which they are a continuation; they contain either air, muco-pus, or gases and pus at the same time. They vary in size from that of a pea to a pigeon's egg. Some authors have described them as bullæ of emphysema (Rilliet and Barthez, Hardy and Béhier, and Vulpian), or as dilated bronchi or bronchial abscesses (Gairdner, Balzer), or as abscesses (West, Damaschino).

C. **Catarrhal pneumonia, epithelial pneumonia, splenitisation.**— Under these names an anatomical state of the lung has been described in which the pulmonary alveoli are more or less completely filled with modified epithelial cells, and with a certain number of white or red blood corpuscles in the midst of a liquid granular exudation. This condition does not differ from inflammatory congestion, except that a much more considerable quantity of epithelial cells and lymph cells is thrown into the pulmonary alveoli. Catarrhal pneumonia assists in the formation of the nuclei of lobular pneumonia just described, and it always exists at the periphery of these nodules, that is to say, around the zone of fibrinous or suppurative pneumonia which is directly in contact with the bronchus; but it almost always exists also in other parts of the lung affected with broncho-pneumonia, more especially in the inferior lobes and at the posterior border of the lung or in the inferior part of its superior lobe, where it may cover more or less extensive areas. The altered parts vary in aspect according to the age of the lesion, so that three degrees may be distinguished in its evolution.

In the **first stage**, the surface of the lung is dark red and slightly tense; its tissue is rather soft, heavier than in the normal condition; and it resists the pressure of the finger; crepitation is absent or very slight; if thrown into water the lung slowly falls

to the bottom or floats midway. On inflating it, it allows a little air to enter, and at certain parts it becomes pink without entirely regaining its normal appearance. On dividing the lung, a muco-purulent and opaque fluid, mixed with bubbles of air, is seen to exude from the small bronchi; and the divided surface becomes covered with a layer of opaque blood-stained fluid. On wiping it, it is seen to be uniformly coloured red, and no granulations can be seen. This appearance resembles so much the tissue of the spleen that the name of splenitisation has been given to it. There is no marked difference between this state and that of pulmonary engorgement, such as exists at the commencement of acute lobular pneumonia, which will be described later on. On microscopical examination of hardened sections, the capillaries which ramify on the alveolar septa are seen to be turgid and to project into the interior of the alveoli. A few tumefied granular epithelial cells, of globular or irregular form, with blunt angles, are seen to be here and there adherent to the walls of the alveoli. Many of these cells are detached and free in the alveolus, where they are sometimes polyhedric, sometimes spherical in form; they contain either one oval nucleus or two or three nuclei. Besides these elements there are red blood corpuscles and a few large granular lymph cells; the alveolar cavity is filled with these elements contained in the midst of a fluid holding fine granules in suspension.

In the **second stage**, the colour of the diseased lung is less red; its tissue is more impermeable to air, and it sinks more quickly in water; it can, however, be slightly dilated by insufflation. On dividing the tissue less liquid can be squeezed out. This liquid is also less blood-stained; it is greyish and mixed with small grumous, opaque particles. On wiping the divided surface, the pulmonary tissue is seen to be of a pinkish colour with grey spots, and, on closer attention, small greyish spots, smaller and less prominent than the granulations of acute lobar pneumonia, may be seen. The bronchi contain a muco-purulent exudation. On microscopical examination of sections of these parts of the lung, the capillaries of the alveoli are observed to be less full of red blood corpuscles than in the preceding case; these elements are also rarer in the interior of the alveolus. This cavity is full of free tumefied epithelial cells, of an irregular polyhedric or spherical shape, often multinucleated and with a granular protoplasm, also a few lymph cells. This is typical of epithelial pneumonia.

A fine reticulum of fibrin is rarely met with in some of the alveoli.

At this stage of catarrhal pneumonia, which lasts for a longer or shorter period, resolution may take place. The epithelial and lymph cells, which have not been expectorated, undergo fatty change, become deformed and broken up, and are absorbed in the form of granules by means of the lymph vessels. The cells become re-attached to the walls of the alveoli, remaining, however, for a time tumefied before they become flattened and return to their normal condition. Catarrhal pneumonia sometimes terminates by suppuration. The divided lung is then seen to be

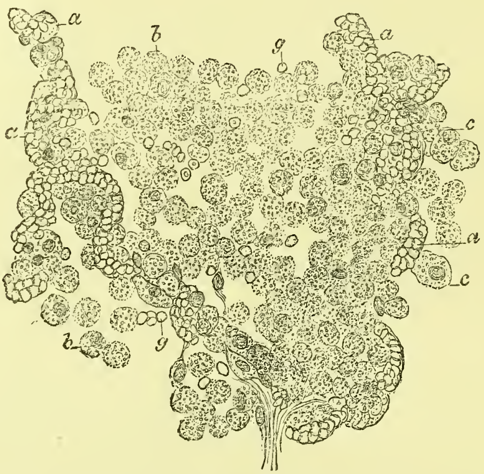


FIG. 46.—SECTION OF A LUNG AFFECTED WITH CATARRHAL PNEUMONIA IN THE FIRST STAGE.

a, *a*, capillary vessels filled with red blood corpuscles and projecting into the pulmonary alveoli; *b*, *b*, lymph cells extravasated into the alveoli; *g*, red blood corpuscles; *c*, *c*, swollen epithelial cells about to be desquamated. Magnified 300 diameters.

covered with a layer of puriform fluid, and the alveoli are filled with lymph cells. Sometimes tracts of catarrhal pneumonia persist for months, and even for a year or longer. They are then either of a yellowish or greyish colour, hepatised and impermeable to air; on division their surface is seen to be of a greyish colour, remarkably dry and very slightly vascular. Nodules of chronic catarrhal pneumonia are quite similar to masses of caseous pneumonia in tuberculosis; they may, however, exist sometimes without tubercles. We have observed a case of this kind in a syphilitic child of three years old, who from the second month had coughed and

shown all the symptoms of broncho-pneumonia. In this case the yellowish and dry lobules showed, under the microscope, alveoli filled with small atrophied cells, which themselves contained fine fat granules ; the capillaries were small or atrophied, contained no red blood corpuscles, and the alveolar septa were not thickened. In the grey or pink lobules, the alveoli contained round or irregularly polyhedric epithelial cells and a few lymph cells. The lobular bronchi were lined with a ciliated epithelium and filled with lymph cells. Around these bronchi a thick zone of newly-formed fibrous vascular tissue was found.

D. **Nodules of fibrinous pneumonia.**—More or less numerous disseminated lobules of broncho-pneumonia are sometimes found, in which the altered tissue has, both to the naked eye and under the microscope, the characters of fibrinous or acute lobar pneumonia. Sometimes also complete hepatisation of the lower part of the pulmonary lobes may exist at the same time. The repair of this lesion is different to that observed in acute lobar fibrinous pneumonia, but the morbid changes are the same. The tissue is impermeable to air, and on cutting the lung across it is seen to have a granite-like appearance with the pink or grey spots of fibrinous pneumonia. Examined under the microscope, the pulmonary alveoli are seen to be distended by a fibrinous exudation, the fibrils of which include lymph cells in their meshes. Further details will be considered when studying fibrinous pneumonia. The course and symptoms of this disease are the same as those of broncho-pneumonia. We have already seen that the connective tissue which accompanies the bronchi and the pulmonary artery into the diseased lobules, and the fibrous septa which circumscribe the alveoli are often very much inflamed and infiltrated with lymph cells in broncho-pneumonia; but the septa of the alveoli are not thickened in this disease, and there is, properly speaking, no interstitial pneumonia. The pleura is always inflamed to a certain degree whenever catarrhal pneumonia spreads immediately under the serous membrane. This pleurisy is generally very slight, without effusion, and is only characterised by a cloudy appearance and slight thickening of the visceral pleura, which is then covered with a very fine layer of fibrin. There are cases, however, notably in the broncho-pneumonia of typhoid fever, in which pleurisy is more acute ; and should the nuclei of suppurative lobular pneumonia be situated under the pleura, they may cause acute and more or less generalised pleurisy.

What distinguishes broncho-pneumonia is, in the first place, that inflammation of the lobular bronchi is the starting-point, and that the oldest and most important inflammatory lesions

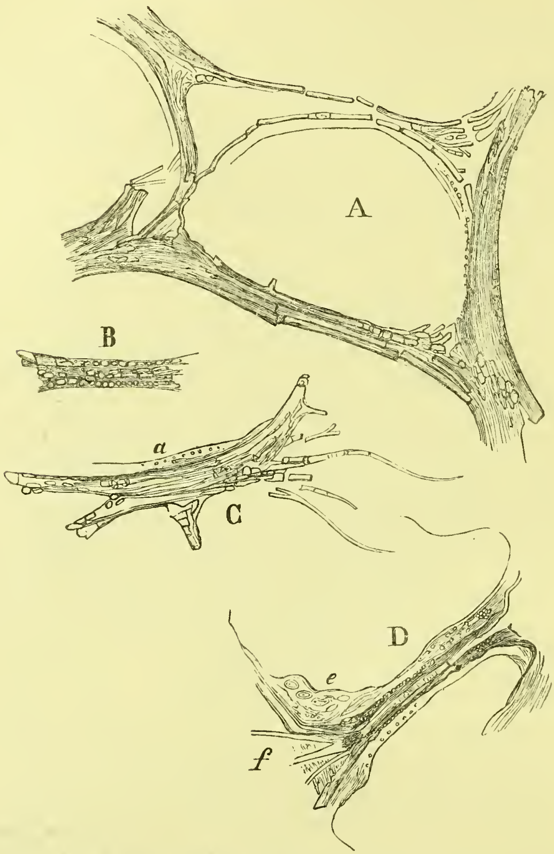


FIG. 47.—ALTERATIONS IN ELASTIC FIBRES, FROM A CASE OF BRONCHO-PNEUMONIA.

A. Elastic fibres bordering a pulmonary alveolus. They have generally preserved the circular form of the alveolus, though they are reduced into small fragments. Magnified 200 diameters.
B. A fragment of this elastic tissue seen under a power of 450 diameters.
C. Appearance given by the small fibres and the large fragmented bundles. *a*, connective tissue of the wall of the alveolus.
D. Fibro-elastic wall which separates two pulmonary alveoli. *f*, elastic fibres; *e*, connective tissue and nuclei belonging to a blood vessel. The connective tissue surrounding the elastic tissue contains fat granules. Magnified 150 diameters.

are seated in and around the walls of these tubes; in the second place, that the multiple lesions, congestion, atalectasia, nuclei of lobular pneumonia, and diffuse catarrhal pneumonia are irregularly distributed, and simultaneously or successively affect

the two lungs. These characteristics constitute a distinct and different species of lobar pneumonia. We may add that broncho-pneumonia often borrows from the specificity of the disease which it complicates quite peculiar characters; as, for example, in broncho-pneumonia consecutive to diphtheria, false diphtheritic membranes, with their fibrinous reticulum, and microspores will be found in the small and large bronchi; in variola, the pustules and false membranes of the bronchi show the structure we have already described; in typhoid fever, caseous or suppurating pulmonary infarctuses are often present, and sometimes pseudo-membranes on the surface of the bronchi. If, in these various diseases, the exudation contained in the pulmonary alveoli be examined under the microscope, micrococci spores will be found, either isolated or united two by two, or in necklace form, often also rod-like bodies in the state of active movement. In infectious diseases, puerperal fever, septicemia, &c., the alveoli contain vibrios, and the lung affected with broncho-pneumonia is often riddled with miliary abscesses. In a case of broncho-pneumonia observed at the autopsy of a syphilitic patient, who died in M. Bouchard's wards at the Charité (1873), we found an alteration of the elastic fibres of the lung. The bundles of elastic fibres were thick, refractile, vitreous, rigid, and broken by a sharp transverse or irregular fracture; they were often dissociated longitudinally, and the isolated fibres showed the same refraction, tumefaction, and facility to break and fragment into molecules. Most of the elastic fibres of the parts of the lung affected with broncho-pneumonia were thus altered. These elastic fibres were not acted upon by any reagent; acetic acid, however, caused them to swell a little, and made the fractured ends approach one another, so that the interstices separating the fragments were less visible. Preparations made after hardening in Müller's fluid, gum and alcohol, showed the same lesions, which in the places where they were most pronounced coincided with the retrogressive period of catarrhal pneumonia, and with partial atrophy of the capillaries.

Clinical varieties of broncho-pneumonia.—We have hitherto studied particularly the various lesions of broncho-pneumonia treated singly. We will now describe the anatomical types according to which these lesions are usually grouped, and which correspond to certain well-determined clinical varieties.

a. The most rapidly fatal form of acute broncho-pneumonia is

suffocating catarrh. Its essential anatomical lesion is capillary bronchitis, that is to say, acute suppurative inflammation of the bronchi of every calibre. At post-mortem examinations there are found, moreover, in the lungs, nuclei of broncho-pneumonia, catarrhal pneumonia at the base of the lungs, atalectasia at their borders, emphysema (which is produced very rapidly), and general acute pulmonary congestion.

b. In another very common type of acute broncho-pneumonia, catarrhal pneumonia predominates. The postero-inferior part of the two inferior lobes of the lung, or these lobes throughout their whole extent, are invaded by catarrhal pneumonia; the apices and the anterior part of the superior lobes are sometimes emphysematous, but in the inferior part of these lobes catarrhal pneumonia alone is present. Collapsed foci of atalectasia are observed on the surface of the lung, in which may also be seen disseminated projecting nodules of broncho-pneumonia. The latter, in various stages of evolution, are present also in those parts of the lung affected with catarrhal pneumonia. On opening the bronchi in the parts of the lung affected with pneumonia they are seen to be very red, covered with muco-pus, and dilated throughout their whole course, particularly if the lesion has lasted some time.

c. Closely related to this form is that in which nuclei of lobular pneumonia predominate. It defines itself; with it may be found all the other lesions of broncho-pneumonia, particularly nodules of lobular pneumonia in various stages of evolution, disseminated irregularly throughout the two lungs, projecting under the pleuræ or deeply hidden in the different lobes, particularly the inferior; this constitutes the essential alteration.

d. At the post-mortem of patients who have died from broncho-pneumonia, nodules of fibrinous pneumonia may be occasionally met with. In the different lobes of the two lungs nuclei of fibrinous pneumonia are found, varying in size from a bean to a nut; these nuclei, in various stages of evolution, and affected with red or grey hepatisation, generally show very evident granulations on section. There often also exists, at the base of one or both lungs, a more or less extensive hepatisation, which may invade the whole of the inferior lobe, and which resembles the second or third stage of acute lobar fibrinous pneumonia. These cases are consequently intermediate between broncho-pneumonia and lobar pneumonia.

e. New-born infants are affected by a special form of pneu-

monia which has been described by some as broncho-pneumonia and by others as lobar pneumonia. It resembles lobar pneumonia in that it generally affects to the same degree one or more entire lobes; but both its naked-eye and microscopical anatomical characters make it difficult to distinguish it from catarrhal pneumonia; hence the reason for mentioning it here. The diseased lobes are dark red on their surface, or marbled grey and red, and are tumefied; on dividing the tissue it is smooth and level in appearance, and a small quantity of opaque blood-stained or greyish fluid can be squeezed out. The pulmonary alveoli are seen, under the microscope, to be filled with cubical or irregularly polyhedric cells. These alveoli have often not been permeated by air, or the respiration has been very imperfect, the pneumonia having commenced almost immediately after birth.

f. Catarrhal pneumonia sometimes takes a subacute or chronic course. The nuclei affected with chronic catarrhal pneumonia are of a grey or yellowish colour; in those which are yellow the circulation is very incomplete, almost nil; the alveoli are filled with tumefied, irregularly polyhedric, cubical or spherical epithelial cells, filled with fat granules. In some cases, but not in all, a very marked dilatation of the bronchi may be observed (Charcot), in which case the peripheral fibrous tissue is thickened; bands of newly-formed fibrous tissue are then found around the lobules and bronchi.

2. **Acute lobar or fibrinous pneumonia.**—Acute lobar or fibrinous pneumonia differs essentially from broncho-pneumonia both by its symptoms and its course. It is a disease cyclic in its course, related perhaps to the presence of microbes; in it the physical signs succeed one another with a regularity foreseen, while the signs of broncho-pneumonia are irregular and changeable. These two diseases differ equally in their pathology. While the lesions of broncho-pneumonia are numerous and various, dissimilar in different cases and proceeding by successive accesses, those of acute pneumonia are always identical and evolve with great rapidity. If the lesions considered in their entirety and grouping are quite characteristic, in each of the two forms of acute pneumonia, the histological process of inflammation of the lungs, consisting in changes in the walls and effusion of an intra-alveolar exudation, does not show much less analogy. Three degrees or anatomical stages may be distinguished in acute lobar or fibrinous

pneumonia. 1st, engorgement; 2nd, red hepatisation; 3rd, grey hepatisation or purulent infiltration.

In the first stage, or that of engorgement, an entire lobe or even a considerable part of two neighbouring lobes are very rapidly affected by an active and very acute congestion. The pulmonary parenchyma is of a brown red; it is distended, hypertrophied, heavier and more compact than in the normal condition; it has lost its elasticity and crepitates slightly on pressure. On cutting into it a sero-blood-stained fluid, slightly aerated, flows out; but portions of the engorged tissue still swim when thrown into water. Congestion and engorgement of the first nucleus of pneumonia are rarely seen in a post-mortem examination; but by auscultation we are enabled to appreciate day by day the progress and growth of the trouble at a certain determined point. If the patient dies during the progress of pneumonia, the anatomical condition of congestion and engorgement can be recognised around the hepatised parts. In sections hardened in Müller's fluid, gum and alcohol, the capillary vessels of the alveolar septa are seen to be turgid and varicose, and project into the alveolus; the alveolar cavities contain red blood corpuscles, a few leucocytes and detached, tumefied, granular, often multi-nucleated epithelial cells. These are the same lesions as those already described in congestion of the lung and catarrhal pneumonia at its commencement. This first period lasts from twenty-four to forty-eight hours.

In the second period, or stage of red hepatisation, the lymph cells and red blood corpuscles continue to be extravasated in large numbers by diapedesis into the interior of the alveoli; the fibrin also escapes from the vessels with these elements and coagulates in the form of filaments around them. Thus is formed a solid exudation which fills and distends all the air cavities, the infundibula, and terminal bronchi. The lung is increased in volume, the ribs leave their imprint on its surface, and it does not collapse on opening the thorax; it is heavy, sinks in water, does not crepitate, and cannot be insufflated; it is very compact and solid in appearance, while in reality it has become very friable. On pressing the finger perpendicularly against its surface, it easily penetrates the tissue. The surface exudes cloudy, grumous, blood-stained fluid, and from the small bronchi whitish clots escape; these are free in their cavity and are composed of fibrin which is not adherent to the mucous membrane. Hepatised pulmonary tissue has a granular appearance when divided, which is still more marked if it be torn; this appearance is due to the

relief formed by the infundibula filled with fibrinous exudation; these infundibula form in fact little masses almost a millimetre in diameter, reddened by the presence of blood. The quantity of blood effused into the lung may be so great, especially in certain animals, as the horse, at the commencement of fibrinous pneumonia, as to give the appearance of apoplexy. On washing the divided surface of the lung by a stream of water to cleanse it from blood, it changes from red to grey or yellowish grey, which colour is caused by the fibrinous exudation containing lymph cells. On scraping the surface of the divided hepatised lung with a scalpel, small grumous, grey, granular particles are obtained; they represent moulds of the alveoli and infundibula (*vide* fig. 48). These coagula may be examined in situ in sections of hepatised

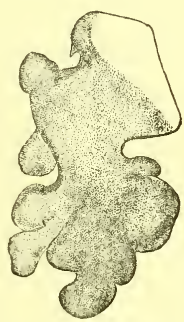


FIG. 48.—FIBRINOUS MOULD OF AN INFUNDIBULUM AND ITS TERMINAL ALVEOLI IN ACUTE PNEUMONIA. Magnified 40 diameters.

lung. Under a low power, the infundibula and alveoli are seen to be filled and distended by an exuded mass, and it may be ascertained that the alveolar walls are not thickened except by the distension of their vessels with blood corpuscles (fig. 49). The exudation of fibrinous pneumonia is composed of a fibrillar reticulum of fibrin, including in its meshes a very small number of modified epithelial cells and a large number of pus cells and red blood corpuscles. At the commencement of the second period of pneumonia, the vessels, distended by blood, project into the alveolar cavity, on the walls of which, and in the depression between the vessels, a few tumefied epithelial cells belonging to the epithelial lining of the alveoli may be seen; but in red hepatisation, which has already existed two or three days, and when the alveoli are distended by the exudation, the blood vessels are most prominent, and contain numerous lymph cells. At this period no

cells resembling epithelial cells can be found either on the surface of the septa or on the vessels; the alveoli only contain lymph cells (*vide* fig. 50). The septa of the alveoli show no changes

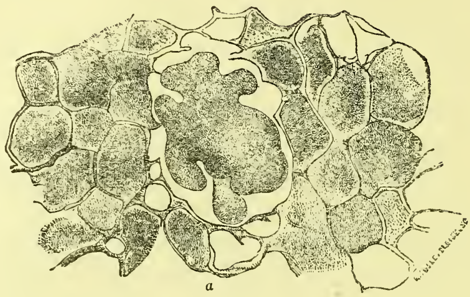


Fig. 49.—Section of a Lung affected by Acute Pneumonia. In the centre of the figure may be seen the mould of an Infundibulum and its Terminal Alveoli. Magnified 40 diameters.

which can explain the friability of the lung. This phenomenon is nevertheless easily understood. Pulmonary tissue is very solid in the normal condition, for it contains a rather considerable quantity of elastic fibres, and on squeezing it between the fingers the air contained in the infundibula is displaced into the bronchi; the

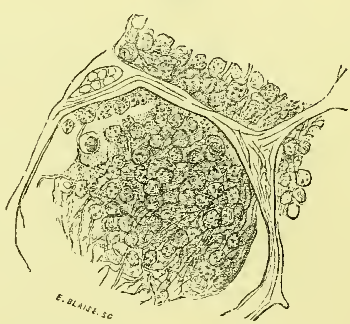


Fig. 50.—Acute or Fibrinous Pneumonia. The Pulmonary Alveoli are filled with Pus Cells entangled in a mass of Filaments of Fibrin. Magnified 200 diameters.

alveolar walls, pressed then close one against the other, constitute a thick and resistent fibro-elastic tissue; but in red hepatisation all the cavities are filled with a semi-fluid friable and fibrinous substance, and on squeezing the tissue between the fingers the exudation cannot be displaced, the distended and delicate walls

of the cavities are ruptured, and the finger penetrates into the hepatised lung with the greatest ease.[1]

The bronchi are red and inflamed; the terminal bronchi, as well as the pulmonary alveoli, are completely filled with a fibrinous exudation adherent to their walls. The lobular bronchi are almost entirely blocked by the same exudation non-adherent to their surface; the bronchi of a medium size contain a transparent viscous fluid holding in suspension fibrinous concretions similar to the preceding, in the form of small cylindrical filaments. These concretions, derived from the small bronchi, are found in the sputa with mucus mixed more or less with blood; on examining these bronchial concretions under the microscope, dichotomous divisions may be recognised, and even the impression of alveolar divisions. The second period of pneumonia, after having lasted from three to five days, terminates by resolution or by passing into suppuration.

When pneumonia terminates by resolution, the fibrin deposited in the pulmonary alveoli loses its fibrillar condition; the network of fibrils is replaced by a fluid in which granules and free lymph cells are suspended, almost all of which contain fine fatty granules. The constituent parts of the exudation, the fibrin and lymph cells, are certainly absorbed almost entirely by the lymphatics; if indeed one considers the large semi-fluid mass, two pints at least, which fills at a certain time the cavities of the lung, and the small amount of the sputa, it is evident that the exudation is not eliminated entirely by expectoration; almost the whole contents of the alveoli are carried away by the lymphatics; we know, moreover, how quickly resolution generally takes place in the acute pneumonia of young subjects. But resolution may

[1] It is important to remark here, that as a direct consequence of the anatomical condition of the lung in red hepatisation, neither insufflation, nor still less physiological inspiration, can make air penetrate into the diseased parts. Hence the abnormal murmurs recognised during life by auscultation cannot be produced either in the alveoli or in the terminal bronchi; nor can the crepitant rale have for its seat either the terminal branches of the bronchi or the infundibula of the hepatised parts, and if it is heard in this stage of pneumonia it is because it is produced in the more or less congested infundibula which are permeable to air near to the hepatised part.* The vesicular murmur is nothing else than the noise of the air passing into the large bronchi, and transmitted to the ear by the hepatised lung; but if the circulation of the air does not take place in the large bronchi connected with that part of the lung affected with pneumonia, and if the greater part of the two lobes or a whole lobe is impermeable, this lung cannot act as a bellows to draw in the external air, and the blowing sound will cease (massive pneumonia of Grancher).

* For further details see Cornil, 'Leçons sur l'Anatomie pathologique et sur les Signes fournis par l'Auscultation dans le Poumon' (*Progrès médical*, 1874).

occur more slowly, and though the general symptoms have subsided, persistent hepatisation may be recognised by auscultation. We have examined the lungs of patients who have died of an intercurrent disease fifteen or twenty days after the cessation of the fever and general symptoms of acute pneumonia, but in whom fine crepitant rales had been heard at a spot over the part originally hepatised. In the post-mortem examination of these cases one or more islets of pulmonary tissue have been found, which are flabby, heavy, impermeable to air, and which blend insensibly with the healthy parts ; isolated from the normal tissue, they sink when immersed in water. On section they show a dark red base scattered over with whitish, yellowish, and opaque spots, which are smaller than the granulations of acute pneumonia.

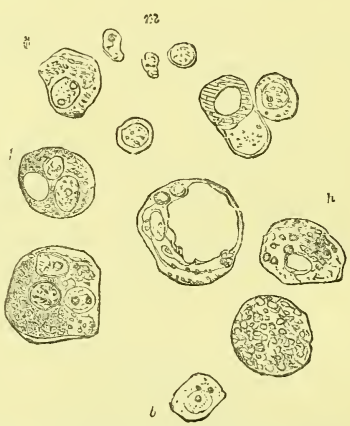


FIG. 51.—ELEMENTS IN A STATE OF GRANULAR DEGENERATION, FROM A CASE OF PNEUMONIA UNDERGOING RESOLUTION.

a, granular body ; *b*, *b*, pavement cells ; *e*, vesicular cells and white blood corpuscles ; *f*, epithelial cell containing two nuclei and showing a cavity ; *m*, granular fragments. Magnified 400 diameters.

On hardening a small portion of a lung which has undergone these changes in osmic acid, and on examining sections stained by carmine under the microscope, all the alveoli are seen to be filled with a fluid coagulated and slightly stained by the osmic acid. In this fluid are found numerous lymph cells, all containing fat granules varying in size and stained black by the osmic acid, and also small round cavities from which lymph cells have fallen during the process of mounting. The alveolar walls show one or more layers of large polyhedric cells, of which those nearest the wall form a true and more or less continuous membrane ; these cells are slightly flattened, with a central swelling and a large ovoid

nucleus ; they are, in fact, the flat epithelium in the act of being reconstituted on the surface of the alveoli, and which remains tumefied till resolution of the products of inflammation is completed (fig. 52). This layer of tumefied epithelium is always observed in slow inflammation of the lung, and at the period of decline of acute inflammation.

If the lymph cells continue to be extravasated into the alveoli, pneumonia, instead of going on to resolution, passes on to purulent infiltration.

In the third stage, or that of grey or purulent infiltration, the surface of the lung is seen on section to be of a pale or yellowish grey colour, and the granular appearance is less marked.

FIG. 52.—WALL AND CONTENTS OF A PULMONARY ALVEOLUS IN THE STAGE OF RESOLUTION IN ACUTE PNEUMONIA.

The preparation was made with osmic acid. The fluid coagulated by the osmic acid in the alveolar cavity contains lymph cells, *a*, filled with fat granules, and red blood corpuscles, *g* ; from the cavities, *b*, lymph cells have fallen out ; *c*, tumefied epithelial cells applied against the alveolar wall ; *c'*, the same cells adherent to that part of the exudation located in the middle of the alveolus. Magnified 250 diameters.

On squeezing the lung a thick reddish-grey pus escapes ; its tissue is very friable, and on pressing it with the finger a hole is made which becomes filled with grumous pus. On microscopical examination the alveoli are seen to be filled with granular lymph cells, which usually contain fine fat granules. The fibrin which formerly existed in the form of fibrils is now almost everywhere amorphous and granular ; the alveolar septa no longer show any trace of epithelial cells ; the capillary vessels of the alveoli contain white blood corpuscles and a few red blood corpuscles, and the alveolar septa are not thickened. Grey hepatisation usually causes death, and it occurs chiefly in old subjects or in persons living under very bad hygienic conditions or who are already much

debilitated. Old subjects often succumb to grey hepatisation five or six days after the commencement of pneumonia.

Acute lobar pneumonia, which almost always affects the surface of the lung, is constantly **complicated with a certain amount of pleurisy.** The visceral pleura becomes covered by a delicate but slightly adherent pseudo-membranous layer, which gives it a shagreen and tomentose appearance. Sometimes the false membranes are thicker and form superimposed layers; they are composed of pus cells, large flat or tumefied endothelial cells, and networks of fibrin; they very quickly become vascular, and on examining them after allowing them to macerate in Müller's fluid, a complicated vascular network may be seen (*vide* vol. i. p. 430). A large fluid effusion is very rarely seen in the pleura; in fact, the abundant formation of fluid characterises acute primary pleurisy. Sometimes it complicates pneumonia when it is said to be pleuro-pneumonia. The delicacy of the visceral pleura, it being hardly ·0050 mm. in thickness, and its circulation being directly dependent on that of the contiguous alveoli, perfectly explains how it becomes inflamed in pneumonia. On the other hand, acute inflammation of the pleura may be communicated to the contiguous pulmonary tissue. This may particularly occur in certain rare cases of pleurisy which cause purulent foci on the surface of a lobe, which cases have been called *dissecting pneumonia.*

In pneumonia, the lymph vessels on the surface of the lung are constantly inflamed and obstructed by an inflammatory exudation. This exudation is the same as that which fills the alveoli. Thus in catarrhal pneumonia the lymphatics contain either tumefied endothelial cells, or lymph cells and fibrin; and in fibrinous pneumonia they contain fibrin, white blood corpuscles, a few red corpuscles, and still fewer endothelial cells. In section of a lung affected with pneumonia it is impossible to say if a certain vacuole filled with a pneumonic exudation represents the transverse section of a lymph vessel or of an alveolus. The wall is alike in both cases; the lymph vessels of the lung have, like the pulmonary alveoli, a network of capillaries immediately under the endothelium, and when inflamed their contents are identical. All the cavities of the lung—alveoli and lymph channels—being inflamed at the same time and filled with the same exudation, the lymphatics do not become considerably enlarged, as occurs when the pulmonary lymphatics are alone inflamed, in which case they can be easily recognised (*vide* p. 114). Though the

lesions of the deeply-placed lymph vessels are recognised with difficulty in catarrhal and fibrinous pneumonia, it is not so with the superficial lymphatics of the pleura. These appear filled and distended with cellular elements in the different layers of the pleura when examined in sections cut perpendicularly to the surface. The pleural connective tissue is then thicker than normally, owing to the presence of lymph cells, tumefied connective-tissue cells, and fluid effused into the connective-tissue spaces. The existence of this superficial lymphangitis, added to our present knowledge of deep pulmonary lymphangitis, lead us to believe that the whole lymphatic network is inflamed in pneu-

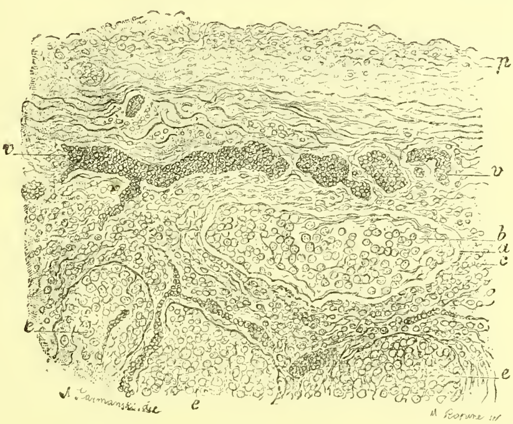


FIG. 53.—SECTION OF THE PLEURA, FROM A CASE OF PNEUMONIA.

A lymphatic is seen, in which the wall, *a*, and its contents, *b*, consist of lymph cells and filaments of fibrin; *p*, pleura; *v*, blood vessels; *c*, *c*, pulmonary alveoli filled with cells. Magnified 100 diameters.

monia. The lymphatic glands at the root of the lung and of the bronchi are also always tumefied, red and inflamed in pneumonia.

Termination and sequelæ.—We have treated hitherto the different stages of acute pneumonia and the lesions of the pleura, blood vessels, and lymphatic glands which result from it; it remains for us now to show how these lesions are repaired. Pneumonia is single or double according as it is limited to one lung or invades both; simple pneumonia most frequently invades the inferior lobe, more rarely the superior. The whole of the diseased lobe may become hepatised, but it is rare for two or three lobes of the same lung to be completely hepatised; some parts still remain permeable to air. Pneumonia does not commence simultaneously in both lungs; when double it generally passes from one to the other, five

or six days after its commencement; in this case it is almost always fatal. Hence it may occur that, many lobes being attacked by acute pneumonia, the disease may manifest itself in some either by grey or red hepatisation, and in others by a more recent red hepatisation, or by engorgement. In lungs affected with emphysema, hepatisation is characterised by the size of the granules, which are nothing else than the large lacunæ of the emphysematous lung filled with a fibrinous exudation. In cardiac cases, pneumonia, whether catarrhal or fibrinous, lobular or lobar, almost always pursues a slower course than acute pneumonia, and is complicated by intense congestion, apoplectic sometimes in character, and often true apoplectiform infarctuses may be produced; this complicated pneumonia is related to blood stasis. Pneumonia in the period of grey hepatisation may sometimes cause abscesses of the lung, which are probably produced by the same process as the peribronchial abscesses of broncho-pneumonia. This is a very exceptional termination. Gangrene following pneumonia is not less rare. In old subjects, in whom pneumonia is particularly serious, suppurative inflammation of the parotid and submaxillary glands may sometimes occur during the period of grey hepatisation. At the autopsy of patients who have died of pneumonia there may be found, besides the lesions of the lung, catarrhal and parenchymatous nephritis, even when the pneumonia has not been treated by frequent blistering, which, as is well known, causes nephritis. The liver is often fatty in these cases. Acute pneumonia is considered by many authors to be a zymotic disease caused by infectious microbes.

Abscess of the lung.—We have already said that abscess of the lung is exceptionally caused by acute pneumonia in the period of grey hepatisation; sometimes a cavity produced by pushing the finger into a hepatised lung, and which becomes filled with puriform fluid and granular débris, has been mistaken for an abscess. Pulmonary abscesses are produced by many septa being destroyed and a communication being established between alveoli filled with pus cells, whence a cavity varying in size with anfractuous walls is formed. Many neighbouring infundibula thus communicate in consequence of the disappearance of their contiguous walls. The abscess increases in size, and if it opens into a bronchus it empties itself and produces a vomica. If the abscess be superficial it may cause perforation of the pleura, and consecutive purulent pleurisy with pneumothorax, or, what is sometimes

observed, the pleuræ become adherent at this point, the intercostal muscles are perforated, and an external fistula is produced. We shall return to this when treating of lesions of the pleura.

Metastatic abscesses of the lung, such as are observed frequently in purulent infection, puerperal fever, typhoid fever, &c., are seen in the form of isolated or confluent nodules. They are characterised, at their commencement, by small nuclei of congestive catarrhal pneumonia, about the size of a pin's head, situated most frequently on the surface of the organ under the pleura. As they increase in size a small drop of pus becomes scarcely perceptible in their centre ; this increases rapidly as the congestion extends towards the periphery and as the nodule of metastatic pneumonia increases. Soon the pus cells, instead of being contained in the infundibulum, are seen to be in the midst of a small purulent focus, resulting from the destruction of the alveolar septa. The pleura shows similar inflammation at the same spot. These nodules of catarrhal or purulent pneumonia, instead of remaining disseminated, often become confluent, thus causing a more extended patch of pleural pneumonia, which is lobulated at its periphery. If the circulation continues to be carried on in the part thus altered an abscess is formed. If the capillary vessels are compressed by the intra-alveolar exudations, blood stasis is produced and consequent thrombosis ; all the diseased part mortifies, and a white infarctus is produced, of irregular form, caseous, whitish in appearance, and surrounded by a highly vascular zone in which diffused hæmorrhages are often observed. Whether these nodules be isolated or confluent, they are always formed, at their commencement, by extravasation into the alveoli of a large number of lymph cells, swollen and proliferating epithelial cells, and free red blood corpuscles. As in the most acute pneumonia, the vessels are filled and distended by blood corpuscles. In the caseous parts of the infarctus the contents of the alveoli are only composed of fat granules and crystals of the fatty acids. The edges of the alveoli can still be recognised by the persistence of the elastic fibres, but the blood vessels are no longer recognisable. At the border of the caseous parts the pulmonary tissue shows the characteristic lesions of catarrhal or suppurative pneumonia or of apoplexy. What is the pathogenesis of the pulmonary lesion in purulent infection ? Is it caused by an embolism, as Virchow thinks, or by inflammation related to another cause ? We were the first (Société Médicale de Lyons, March 1871) to call attention to the fact that the

lesions of purulent infection could not be attributed to simple embolism, but that they were probably due to a ferment or microbe which, after circulating in the blood vessels or lymphatics, causes a local irritation in the organs. This view was developed, almost simultaneously, by Klebs and Recklinghausen. The pneumonia of *glanders* is a suppurative pneumonia in which the lesions resemble those of metastatic foci.

Inflammation of the lymph vessels of the lung.—We have already seen that the lymph vessels of the lung are constantly inflamed in broncho-pneumonia and lobar pneumonia, and that they become filled with an exudation similar to that in the pulmonary alveoli. Here we may observe, as in pneumonia: 1st, an inflammation similar to that of catarrhal pneumonia in which tumefaction and multiplication of the endothelial cells lining the lymphatics are present. They are then distended by these elements mixed with lymph cells. Sometimes filaments of fibrin are present in the fluid. Secondly, a fibrinous inflammation, in which the vessels are filled with lymph cells and fibrin; and thirdly, a purulent inflammation, in which the fluid contained in the vessels has the naked-eye characters of pus and contains a considerable number of lymph cells. This purulent inflammation of the lymph vessels, which is met with in puerperal fever, and infectious diseases generally, coincides with purulent pleurisy and abscesses of the lung. In lymphangitis, the lymphatics of the lung always contain a rather considerable number of red blood corpuscles, which is due to the fact that the blood capillaries are in immediate contact with the endothelial wall of the lymphatics, a condition very favourable to diapedesis.

We have already described the various forms of pulmonary lymphangitis in considering pneumonia. We will also return to them in tuberculosis. It may be said, in a general way, that the pulmonary lymphatics show the same lesions as the alveoli, with which they communicate. Pulmonary apoplexy is in fact accompanied, as we have seen, with effusion of blood into the lymphatics of the lung. Inflammation of the superficial and deep lymph vessels of the lung rarely occurs independently of pneumonia and pleurisy. A certain number of cases have, however, been observed and published by Raynaud, Féréol and Thaon, Troisier and ourselves, in which lymphangitis, consecutive to cancer of the stomach, lymphadenia, syphilitic lesions of the stomach and liver, has caused lesions in the bronchial glands, which latter were

particularly remarkable for the enormous distension of their lymph vessels, and by the caseous condition of the centre of the exudation which filled them. On the surface of the lung the interlobular networks of lymphatics form moniliform chains, from $\frac{1}{2}$ to 1 or 2 mm. in diameter, and are filled with whitish or yellowish material; they become larger as the root of the lung is approached. They are seen in a section of the lung along the bronchi and pulmonary arteries. Examined in the fresh state, their contents generally show two layers, one attached to the wall of the vessel and composed of numerous swollen endothelial cells, polygonal in shape with blunt or rounded angles measuring from 10 μ to 18 μ in diameter, and composed of a granular protoplasm; they have no limiting membrane and contain large oval or spherical nuclei. The other is at the centre of the lumen of the vessel; it is composed of a yellowish and opaque caseous coagulum, formed of granulo-fatty lymph cells. Just as lymph vessels contained within pulmonary tissue affected with pneumonia become inflamed secondarily, so do bronchi and pulmonary alveoli near the lymph channels primarily inflamed show in their turn the lesions of bronchitis and catarrhal pneumonia. Thus, in a case of pulmonary lymphangitis which we observed consequent on syphilitic tumours of the stomach, we found that in transverse sections of the peribronchial lymphatics the adjacent bronchus was always seen to be filled with lymph cells, at the same time that the neighbouring alveoli were full of desquamated epithelial cells and lymph cells, as in catarrhal pneumonia. Moreover, in these lymphangites with caseous contents it is not unusual to find cancerous lymphangitis coincident with carcinomatous nodules in the lung, and a similar degeneration of the bronchial glands. We shall describe these lesions of the lymphatics with carcinoma of the pleura, a disease in which they are easily observed.

Gangrene.—Pulmonary gangrene is sometimes the result of pneumonia, or of pulmonary hæmorrhage; it is more frequently related to obliteration of the pulmonary or bronchial arteries, or it may be caused by infectious diseases, typhoid fever, measles, carbuncle, &c., or by a wound, perforation of the lung, as in rupture of the œsophagus, abscess of the mediastinum, &c. Gangrene of the lung may occur in two anatomical forms: it may be circumscribed or diffused.

1. **In circumscribed gangrene** there are usually many disseminated foci in one or in both lungs; the gangrenous and

softened part is always at the centre of a patch of lobular or catarrhal pneumonia. There is no doubt that these patches of pneumonia usually precede the gangrene which occurs at their centre; in fact, in many cases we have seen patches of lobular pneumonia some of which already contained a small gangrenous focus, while others were free. We also believe that the foci of gangrene which are observed in a certain number of infectious diseases are the result of broncho-pneumonia, which shows, in consequence of the general condition of the patient, or from special

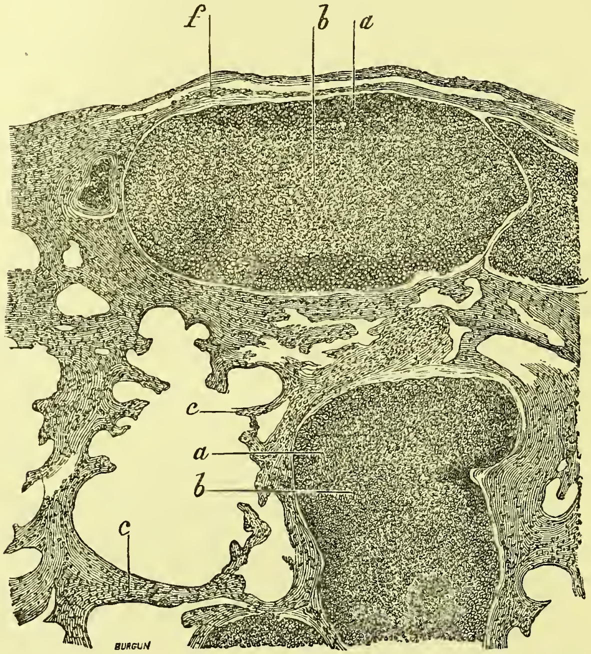


Fig. 54.—Inflamed Lymph Vessels of the Lung seen in Section.

f, pleura; *a*, peripheral layer of the exudation which fills the lymphatics; this layer is formed of large endothelial cells; *b*, central contents of the lymphatic composed of caseous lymph cells; *c*, *c*, septa of the pulmonary alveoli. Magnified 40 diameters.

causes, a tendency to terminate by gangrene. Each one of these patches of lobular pneumonia terminating by gangrene shows at the centre a more or less extensive anfractuous cavity, in the walls of which is seen a slate-coloured débris. If the vomica is rather large, projecting and detached vessels are often remarked on its surface. It is filled with a grumous substance, more or less fluid, and grey in colour, and it communicates with a bronchus. Both cavity and fluid exhale a peculiar and fœtid cadaveric odour.

On dividing one of these indurated nodules, three distinct layers are seen in section : the first is formed of greyish débris, or of a pulpy adherent mass which limits the loss of substance, beneath which the wall of the cavity appears of a deep red colour ; the second is composed of hepatised, greyish, friable pulmonary tissue. In both these layers all the vessels of whatever calibre, arteries, veins, and capillaries, are obstructed by a fibrinous clot. The third layer, which is continuous with the surrounding healthy parts, generally shows the lesions of catarrhal pneumonia in the second stage, and sometimes those of fibrinous pneumonia. In the peripheral zone the lesions of pneumonia are discovered by microscopical examination ; the alveoli are filled with pus cells, and the capillaries are distended with blood. The grey or hepatised intermediate zone shows the histological characters which are always present in pulmonary hepatisation before ending in eliminative gangrene. This is, properly speaking, gangrenous pneumonia or mortification of the hepatised lung preceding cadaveric putrefaction.

This tissue, absolutely deprived of air, is grey in colour, slightly transparent, and on looking at it closely branching lines and yellowish opaque spots are seen, the colour and opacity of which are due to the nature of the exudation which fills the terminal bronchi and alveoli; in fact, in sections of the tissue large spherical cells measuring 15 μ to 20 μ, and filled with fat granules, are found in the midst of the fluid in the alveoli, which also contains lymph cells. These granular bodies are generally nucleated, and it is they which give the contents of the alveoli and small bronchi their opacity and yellowish colour. In the fluid and cells obtained by scraping, a large quantity of micrococci are present. The vessels are filled with coagulated fibrin. Tissue thus hepatised is very friable and gorged with fluid. This lesion is met with in almost all forms of pulmonary gangrene at the edge of the part undergoing putrefaction, and it may also often be observed in the pneumonia of tubercular subjects when about to terminate by ulceration. The centre of patches of gangrenous pneumonia which have not yet ulcerated is changed in the same manner. The solid greyish débris of the putrefied pulmonary tissue which lines the ulcerating cavity is composed of blood vessels and elastic fibres which are more or less adherent to the preceding zone. Thus, in sections passing through the internal and middle zones, the débris of elastic fibres and vessels is seen to be continuous with the same elements of the hepatised parts. In sections

stained with methyl violet it may be ascertained that the alveoli in the two internal zones of the gangrenous cavities contain a large quantity of micrococci. In this form of gangrene the putrefaction and molecular destruction commence at the spot where the necrosed tissue comes in contact with the external air; that is to say, where the bronchus pierces the centre of the lobule. The destruction proceeds from point to point, and the products of cadaveric decomposition and the fluid remain for a longer or shorter time in the ulcerating cavity before being expectorated. This fluid contains pus cells and large cells infiltrated with fat granules; grumous particles composed of filaments of connective or elastic tissue; black, yellow, or orange pigment granules derived from the colouring matter of the blood; crystals of ammoniaco-magnesian phosphate, margarin, leucin, and tyrosin; and, finally, schyzomycetes. Fungi resembling leptothrix buccalis may also be met with. All these elements are found in the sputa, which have a characteristic odour, and are generally greyish in colour, slightly blood-stained, and puriform; mixed with water they separate into three layers like the sputa from bronchiectasic cavities. If gangrene affects the peripheral lobules of the lung, it causes fibrinous pleurisy at that spot, and on the gangrenous cavity increasing in size it sometimes opens into the pleura, producing pyo-pneumothorax. In this form of gangrene the fibrinous clots which fill the blood vessels are found also in all the part affected with what we have called gangrenous pneumonia, but are scarcely ever found beyond.

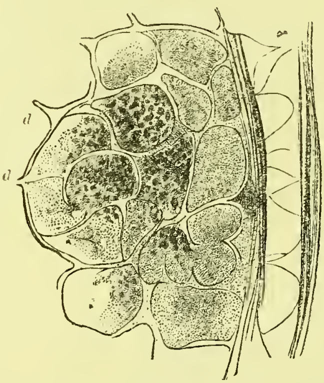


FIG. 55.—SECTION OF A LUNG AFFECTED WITH TUBERCULAR PNEUMONIA.

In the group of alveoli, *d*, *d*, are seen opaque spots produced by the granular bodies. Magnified 30 diameters.

2. **Diffused gangrene.**—Diffused gangrene may occur at the end of acute fibrinous pneumonia in the third stage. In the horse it is a frequent termination of pneumonia; the gangrene is then caused by coagulation of fibrin in the blood vessels. In man this form of gangrene is sometimes caused by the embolic obstruction of a large branch of the pulmonary artery. The mortified part of the lung is more external and more irregular in form than in circumscribed gangrene, but the details, the course, and the result are the same. We have seen a case in which the hepatised and mortified tissue was quite in the first stage of putrefaction. It was a case of acute pneumonia ending in suppuration. The mortified part was greyish, anæmic, and infiltrated, extremely friable, and at a certain spot there was a slate-grey and fœtid part which was not yet completely separated from the peripheral tissue. The ulcerative cavities caused by this form of gangrene are anfractuous, very extensive, crossed by vascular bridges, and are filled with an ichorous, serous or puriform fluid. When the gangrene is located at the periphery of the lung, immediately under the pleura, pyo-pneumothorax is rapidly produced.

Interstitial pneumonia.—Under the name of interstitial pneumonia we intend to describe many states of the lung, which differ greatly in their cause, and which are far from being the same from the anatomical point of view, but which may be grouped together by the fact that they present one common characteristic, namely, inflammatory thickening of the fibrous framework of the lung. Multiplication of the connective-tissue elements of the pulmonary septa is the characteristic of interstitial pneumonia. Slowly developed, it constitutes a distinct variety, chronic pneumonia. The indurated and thickened pulmonary tissue is generally pigmented and of a black or slate-grey colour. Interstitial pneumonia is partial when it is caused by a limited lesion, as, for example, by old vomicæ, miliary tubercle, dilated bronchi, abscesses which have undergone resolution, chronic pleurisy, &c. It affects a whole lobe or may be general when it is due to acute pneumonia, which is very rare, or to penetration into the lung of coal dust, atoms of flint, steel, &c.

As in these various forms of interstitial pneumonia the anatomical phenomena which accompany thickening of the septa are not the same, we shall proceed to describe them separately. Fibrous induration of the lung is a constant phenomenon. The septa of the alveoli are very thick, hard, and fibrous in appearance.

On examining sections of them under the microscope the septa are seen to contain a large number of small cells. At the beginning of the morbid process these cells are round, but later on they become slightly elongated and flattened, and located between fasciculi of newly-formed connective tissue. The lumen of the alveoli, diminished at first, finally disappears entirely, when the walls, greatly thickened, meet together ; and thus fibrous transformation of an entire portion of a lung may be produced. This may be particularly observed in certain cases of chronic pleurisy

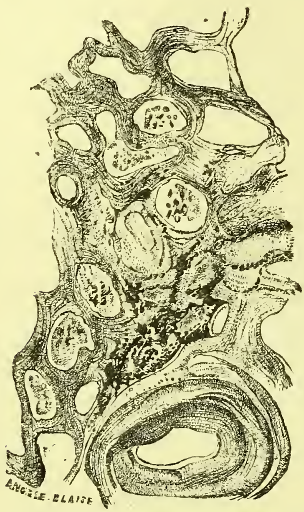


Fig. 56.—Section of a Lung affected with Interstitial Pneumonia.

The thickened septa of the alveoli are pigmented, particularly around a vessel at the lower part of the figure. The alveolar cavities contain round and pigmented cells. Magnified 30 diameters.

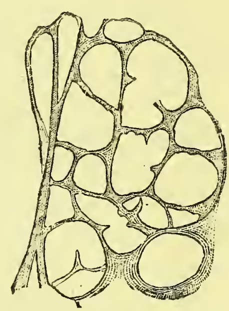


Fig. 57.—Section of the Lung in the Normal Condition, to show the thickness of the Alveolar Septa compared with that of the adjacent Figure. Magnified 40 diameters.

which have caused considerable fibrous thickening of the visceral layer of the pleura. Then the cortical part of the lung, particularly at the apex, undergoes the same change ; it cannot collapse, it creaks under the knife, and shows both to sight and touch all the characteristics of fibrous tissue. Under the microscope the connective tissue of the alveoli shows a more or less marked black pigmentation, particularly around the blood vessels, the walls of which are much thickened and are continuous with the neighbouring fibrous tissue. The arteries, which are often flexuous, remain gaping when cut across.

a. **In old subjects** there is a lesion of the lungs which consists in a **slate-coloured induration** of the apices; this condition is so frequent that it might be almost regarded as physiological. The tissue of the morbid parts is hard, elastic, blackish, and does not crepitate; at the same spot depressed cicatrices are often observed, or callosities of the pleura and dense fibrous pleural adhesions. In sections, the pulmonary tissue seems to be composed of very much thickened septa, which limit retracted alveoli, or, on the contrary, of emphysematous dilatations surrounded by dense fibrous tissue infiltrated with black pigment. Often also in the midst of the indurated spots caseous or calcareous nodules are found, located in small round cystic or cylindrical cavities. These are often closed at both ends, but their continuance into a bronchus can sometimes be traced. These cavities, containing a caseous or cretaceous substance, which is nothing else than altered pus, have been considered by some authors to be tubercles which have undergone cure; but if this is sometimes true, it cannot be doubted that they are also the remains of other old morbid processes, such as bronchial dilatations, pulmonary abscesses, infarctuses, &c. In this form of interstitial pneumonia the needles and small bony masses described in vol. i. p. 229, are sometimes met with in the apices of the lung.

b. **Syphilitic pneumonia.**—In this variety, which is found almost solely in new-born children, the diseased part of the lung shows no trace of pigmentation; hence the name of *pneumonia alba*, which has been given it by Virchow. The cellular elements contained in the interalveolar septa are round and embryonic. The septa are extremely thick. The alveoli, though greatly diminished in volume, are sometimes still permeable to air; their internal walls are lined with pavement epithelium, the cells of which, becoming free in the interior of the alveolus, are spherical and infiltrated with fat granules (*vide* fig. 59). The density and resistance to pressure are first observed in the diseased part, but on dividing the tissue a white or greyish tissue is seen, fibrous in appearance, and difficult to tear or indent with the finger nail. These nuclei of interstitial pneumonia may become true syphilitic gummata projecting on the surface of the lung. In some cases they cause bronchitis and catarrhal pneumonia around themselves. This interstitial syphilitic pneumonia, accompanied with the formation of gummata, must be distinguished from catarrhal pneumonia or chronic broncho-pneumonia, sometimes observed in older syphilitic children. The latter does

not differ from chronic broncho-pneumonia, described above. Interstitial pneumonia and syphilitic gummata are extremely rare in adults.

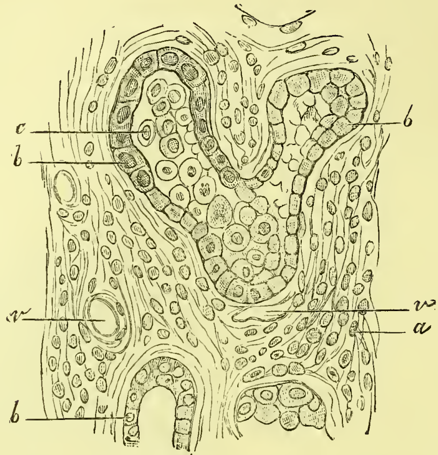


FIG. 58.—SECTION THROUGH THE NUCLEUS OF HEPATISATION, WHICH IS REPRESENTED BELOW.

a, connective tissue of the lung proliferating ; *b*, pavement cells lining the alveoli ; *c*, round cells free in the interior ; *v*, vessels. Magnified 300 diameters.

c. In repeated congestion of the lung following hæmorrhagic infarctuses, in a peculiar form of miliary phthisis, and above all in chronic cardiac disease, the lung often shows indurated and

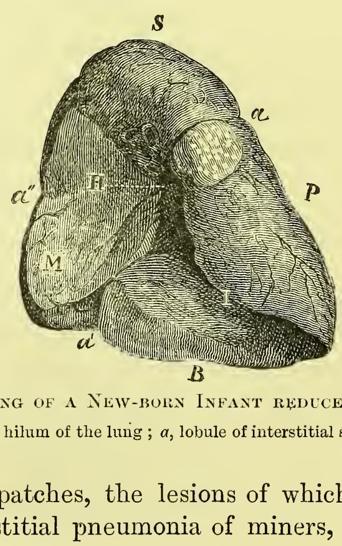


FIG. 59.—LUNG OF A NEW-BORN INFANT REDUCED ONE-THIRD.

S, apex ; B, base ; H, hilum of the lung ; *a*, lobule of interstitial syphilitic pneumonia.

black pigmented patches, the lesions of which are the same as those of the interstitial pneumonia of miners, or anthracosis.

d. **Pneumonokoniosis.**—This name is given to diseases of the lung caused by the inhalation of various kinds of dust. The most common is **anthracosis**, or the accumulation of carbon in the lung. The lungs of adults or old persons often contain granules of carbon which are deposited in the blood vessels and the interlobular lymphatics. This distribution of the particles of carbon is easily recognised on examining the surface of the lung. On inspiring air containing smoke, molecules of carbon are drawn into the air passages; if they do not penetrate as far as the alveoli, they are

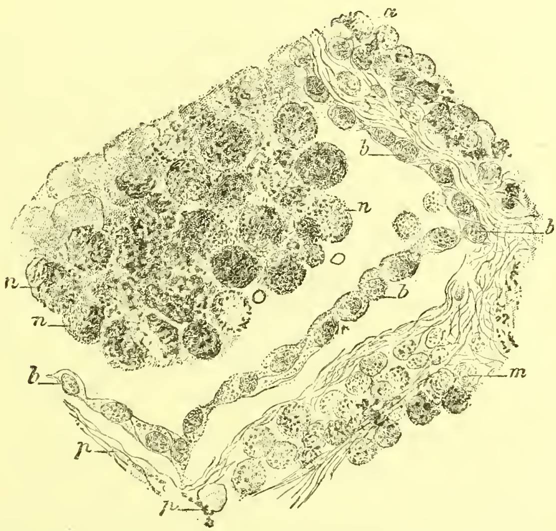


Fig. 60.—Section of a Lung affected with Interstitial Pneumonia, with Pigmentation of the Cells.

a, *m*, pigmented lymph cells located in the alveolar septa; *b*, *b*, *b*, tumefied epithelial cells forming a delicate membrane on the surface of the alveolar wall, from which it is partly detached; *n*, *n*, *n*, round pigmented cells in the centre of the alveolar cavity. Magnified 300 diameters.

carried on by the cilia lining the bronchi and are cast out with the sputa; but if some of these particles of carbon become deposited in the alveoli, they are either lodged in the fibrous septa of the lung, or are taken up by the lymph cells and are carried away into the pulmonary connective tissue around the vessels, or into the lymphatic glands; whence results pigmentation, varying in depth, of the lung and bronchial glands. But if workmen live in an atmosphere constantly charged with particles of carbon, such as charcoal burners, colliers, copper founders, &c., they suffer first from bronchitis, then from a peculiar form of pneumonia, which

terminates by ulceration and the formation of cavities. A portion of the lung becomes altered; the diseased parts are dense, slate-coloured, or black; they generally project from the surface of the lung, which seems hypertrophied at this spot. On dividing the organ the surface of the indurated parts is seen to be smooth, hard, regular, slate-coloured, or black, or of a brilliant ebony black if the lesion is very marked; the tissue is so hard that the finger nail can make no impression, and the finger is stained black. On scraping with a scalpel a thick black fluid is obtained. The bronchi contain a blackish muco-pus, and the sputa have the same appearance.

In sections examined under the microscope the interalveolar septa are seen to be very thick and infiltrated with black molecules; these are located in the external wall of the vessels, in the protoplasm of the cells, and between the fibres of the connective tissue. Inside the alveoli, which are more or less contracted, round cells are found about the size of pus cells or larger, and containing black granules. Lining the alveolar septa a layer of large epithelial cells is generally seen; they are tumefied and contain one large oval nucleus and are often partly detached. They adhere together in such a way as to form a kind of floating membrane (*vide* fig. 60). In the lumen of the alveoli small corpuscles of carbon are found; these are free and are agitated by Brownian movements; they are generally irregular and angular in shape, but are sometimes round; in colour they are absolutely black, but particles of wood carbon have, however, a ruby red colour when not too many are massed together (Rindfleisch). These particles of carbon have been introduced by way of the larynx, trachea, and bronchi. The observations made by Traube (quoted on p. 82), and in which Rindfleisch made the microscopical examinations, allow of no doubt on this point. These particles do not penetrate across the layer of cylindrical ciliated epithelium which lines the air passages, but when in the lung they easily, by reason of the irritation they produce, cause desquamation of the delicate epithelium of the alveolus, and so become encrusted in the loose connective tissue of the septa. The pus cells seize on them, as they do all kinds of fine dust, and they are expectorated with the sputa.

These particles of carbon are carried by a different route and by the same elements into the circulation of the lymph and into the bronchial glands; the mesenteric glands are also impregnated with them, and become considerably enlarged. At the last stage

of the disease the black and indurated parts of the lung ulcerate at their centre, and cavities are formed similar to those of pulmonary phthisis, with this difference, that there are no tubercles, and that the indurated tissue which forms their walls and the pus which they contain are of a black colour. Workmen who work with iron or steel are subject to a similar pneumonia (siderosis), but in which the discolouration is brown instead of black. Workmen exposed to flint dust are also attacked by a similar interstitial pneumonia, caused by the penetration of silex, which fact has been proved by Kussmaul and Schmidt by means of chemical analysis. Besides the bronchitis and interstitial pneumonia directly caused by the presence of foreign particles in the

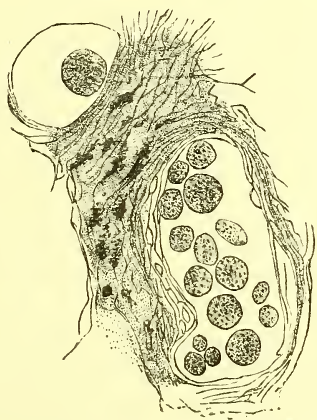


FIG. 61.—SECTION OF A LUNG AFFECTED WITH INTERSTITIAL PNEUMONIA.

In the alveolar septa black pigmented connective tissue cells are seen, and in the interior of the alveolus round cells equally pigmented. Magnified 200 diameters.

lungs, workmen who are exposed in their work to any kind of dust are predisposed to tuberculosis, and are often affected by it.

e. **Lobar pneumonia which has passed into the chronic condition** is extremely rare, but is sometimes met with in infirmaries where old persons are treated. Charcot[1] has described three forms distinguished by their colour: red, grey, and yellow hepatisation. It must not be thought that these forms are similar to those of acute pneumonia, or that there is between them a constant relation of succession. We believe that the difference of colour is due to the fact that some of the alveoli contain blood or the pigments which result from changes in colouring matter of the

[1] *Thèse d'Agrégation*, 1860.

blood, while the grey or yellow colour is due to an abundance of fat granules. In these cases of chronic pneumonia the inter-alveolar septa are thickened, and are more or less infiltrated with black pigment derived from the blood; the alveoli are filled with large round cells, pigmented or fatty-granular, with lymph cells and sometimes red blood corpuscles. In some cases the predominating elements found in the alveoli have been large corpuscles filled with fat granules. Cases of this disease have also been recorded in which cavities were formed. It is necessary to subject a lung to a very profound study before making the anatomical diagnosis of chronic pneumonia, and tuberculosis cannot be eliminated till after a thorough examination. In every case of interstitial pneumonia which we have examined, the lesion, when situated on the surface of the lung, is accompanied with chronic pleurisy characterised by considerable fibrous thickening. The limits of the indurated pleura and pulmonary tissue are well marked by the pigmentation of the tissue, and by the arrangement of the alveolar vessels, even when the alveoli are contracted or effaced by a new formation of connective tissue.

Tumours of the lung.—Almost all kinds of tumours have been observed in the lungs, but the most common and important are tubercle.

Sarcoma has been observed in the lung only in the form of secondary nodules following primary tumours in another part of the body, such as the bones, the testicle, the breast, &c. In these secondary growths the variety of sarcoma of the primary tumour is reproduced. They develop either in the interior of the alveoli or in the connective tissue of the pulmonary septa. The sarcomatous nodule is composed of a group of alveoli filled with round or fusiform embryonic cells. In the midst of the morbid mass the infundibula and alveolar septa, characterised by the arrangement of their elastic fibres, can be recognised under the microscope. In melanic sarcoma the elements of the new tissue are infiltrated with black or brown granules.

Simple melanic tumours (*vide* vol. i. p. 315), secondary in the lungs, resemble in every respect, both in their naked-eye and microscopic appearances, the interstitial pneumonia of miners, excepting that the black granules are round and fine instead of being angular. A melanic tumour of the lung may invade the bones of the vertebra, and may destroy one or more vertebræ, so as to give rise to a variety of Pott's disease.

Fibroma of the lung has been observed by Rokitansky in the form of small masses, more or less hard, and of the size of a pea or nut. The same pathologist had seen **lipomata** of the size of a lentil or pea, seated under the visceral pleura.

Osteomata (*vide* vol. i. p. 225) are met with in lungs affected with interstitial pneumonia. We have seen a case of osteoïd tumour of the lung characterised by transformation of the alveolar septa into osteoïd tissue (vol. i. p. 222).

Chondromata have been observed in the lung following chondroma developed in another organ.

Primary carcinoma of the lung is rather rare; it is most frequently of the soft or encephaloid variety, and is more often situated in the right than in the left lung; but it often attacks both lungs one after another. It commences in nodules which increase in size and form one or more considerable masses which occupy the largest part of one or many lobes. The visceral pleura always shows considerable thickening at this spot, due to carcinomatous change. In a section of the diseased part, whitish islets or grains are generally seen similar to those of hepatisation; they measure from 1 mm. to 1·5 mm. across, and are separated by septa of pulmonary tissue which are often pigmented. These spots or islets are caused by the filling of the infundibula with carcinoma cells. On scraping the divided surface with a scalpel, these can be scraped out and a milky juice obtained. On examining hardened sections under the microscope the alveoli are found to be filled with large cells which are spherical or polygonal in shape, and usually contain ovoid nuclei and large nucleoli, in one word, carcinoma cells. The alveolar walls are generally intact, exactly as in the normal condition, or they may be slightly thickened by the formation of small round cells between their fibres; their vessels are gorged with blood. There is hence no newly-formed stroma in carcinoma of the lung, for the fibrous framework of the organ takes its place. Secondary cancerous nuclei of the lung following primary carcinoma of the breast, uterus, or any other organ, show the same structure, the elements of the cancer juice filling the alveoli, the septa of which are normal or thick. Primary carcinoma may cause ulceration and the formation of cavities which are sometimes multiple, and which always communicate directly with the bronchi. Hæmoptyses then occur, and the patients expectorate the disintegrated elements of the cavity, that is to say, the cancer juice and the débris of the elastic fibres of the septa. Secondary nodules of pulmonary car-

cinoma may belong to the scirrhous, colloid, hematoid, or melanic variety, and in each the character of the primary tumour is reproduced. The scirrhous nodules are small, hard, and extremely numerous when they follow, as is frequent, old scirrhus of the breast. We have already spoken of this eruption on the mucous membrane of the air passages ; it is continued into the lung itself and on the pleura. The alveoli are in this case filled with smaller elements than in encephaloid. Colloid carcinoma, which is rather frequent in the lung following a primary tumour of the same kind developed in the mucous membrane of the digestive tract and the biliary ducts, is seen in the form of small transparent

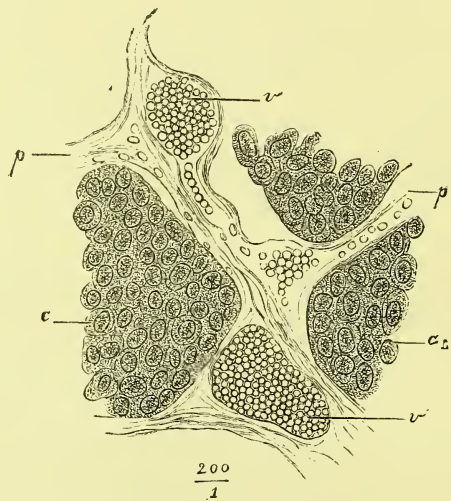


FIG. 62.—CARCINOMA OF THE LUNG DEVELOPED IN THE PULMONARY ALVEOLI.

p, the alveolar septa ; *v*, blood vessels extremely dilated and containing red blood corpuscles ; *c*, carcinoma cells. Magnified 200 diameters.

granules limited by the walls of the pulmonary infundibula. These granules unite to form small spherical nodules ; here again the stroma of the tumours is reduced to the fibro-elastic tissue of the lung. Carcinoma of the lung developes, then, in a markedly different manner from the carcinomata already described (*vide* vol. i. p. 176), of which connective tissue is the type. In the lung, in fact, it is difficult to suppose that the epithelial cells and white blood corpuscles extravasated from the blood vessels are not exclusively the origin of the cells found in the alveoli, and which constitute the whole neoplasm. In a certain number of cases of secondary carcinoma of the lung we have ascertained a

very active participation of the superficial and deep lymphatic vessels in the neoplasm, and a carcinomatous change in the walls and contents of these vessels. (*Vide* cancerous granulations of the pleura.)

Tuberculosis of the Lung.

Tuberculosis of the lung was not studied scientifically till the commencement of this century; before that time the word phthisis was synonymous with consumption. To Bayle we owe the distinction between the various kinds of phthisis, tubercular phthisis and granular phthisis. Then Laennec, in his admirable 'Traité de l'Auscultation Médiate,' described the naked-eye appearances of all the pulmonary lesions of phthisis, among which were included the granulations of Bayle. According to Laennec the tubercular substance is an unique product in the body, a parasitic formation found in the lung in the form either of isolated bodies, or as an infiltration. The former are miliary tubercle, crude tubercle, tubercular granulations, and encysted tubercle. Laennec described three varieties of tubercular infiltration—amorphous, grey, and yellow tubercular infiltration; these he considered to represent various stages in the development of tubercle. This general conception of pulmonary tubercle was held without dispute till Reinhart and Virchow made their researches. According to these authors, the infiltrating tubercle of Laennec, his crude tubercle, and even miliary tubercle are nothing else than pneumonia in which the intra-alveolar exudation has become caseous. Virchow gave a new description of the tubercular granulation; he made it the unique lesion of tuberculosis, and referred caseous pneumonia to scrofula. Buhl and Niemeyer, in insisting still more on this dualism in phthisis, came almost to deny the existence of tubercular granulations in the lung, and considered caseous pneumonia to be the result of acute pneumonia or pulmonary hæmorrhage. They considered (*vide* vol. i. p. 201) tubercles to result from infection following the destruction of caseous foci, an opinion which cannot bear examination. In the treatise which one of us published in collaboration with Hérard, we, while giving a different description of the granulation, and the various kinds of tubercular and caseous pneumonia in which granulations are found, referred nevertheless all these lesions to the same general disease. The German school has, with Virchow, described as pneumonic every

lesion characterised by accumulation of cells in the pulmonary alveoli. This definition being accepted, every granulation primarily developed in the alveoli, or occupying the alveolar cavities, is pneumonic. We have long contended against this point of view, and in the first edition of this manual (1869) we described tubercular granulations originating in the pulmonary alveoli, occupying the infundibula and filling a patch of the lung with small cells. We compared this mode of development with that of tubercular granulations of the thyroid gland in which the closed cavities are invaded (*vide* vol. i. p. 206). The alveoli of the thyroid gland become filled with small cells, which atrophy and become caseous as the centre of the tubercle is reached. The accumulation of the cells of a tubercular granulation in the alveoli of the lung should not surprise us more than to see them in the interstices of the fibres of connective tissue, or in the meshes of the omentum. The day that it is satisfactorily demonstrated that tubercle is developed in the alveolar cavities, and affects the same location and at its commencement the same structure as pneumonia, the scrofulous pneumonia of Virchow will lose the ground regained by tubercle.

Another very important fact towards the comprehension of pulmonary tubercle is the recognition of agminated tubercle, masses formed by the agglomeration of a more or less considerable number of granulations which become confluent (*vide* vol. i. p. 207). One of us described in 1867 [1] confluent granulations in tubercle of bone, and we pointed out in the first edition of this manual that confluent granulations develop and degenerate like discrete granulations. Thaon put forward the same view in his excellent thesis on the pathology of tuberculosis. As many observers, particularly in Germany, had hitherto described the masses formed of confluent granulations under the name of caseous pneumonia, the field of caseous pneumonia was still further diminished. It should be stated, moreover, that one of us,[2] in studying the mode of development of tubercle described by Virchow, along the vessels of the pia mater remarked a similar tendency to the development of tubercular tissue around the vessels of the lung and around the bronchi. We considered that the lesion there described under the name of peribronchitis is a tuber-

[1] Ranvier, 'Tubercules des Os,' *Arch. de Phys.* 1868.

[2] Cornil, 'Du Tubercule spécialement étudié dans ses rapports avec les Vaisseaux,' *Arch. de Phys.* 1868.

cular change; this again limited the region of simple inflammation in favour of tubercle. The excellent works of Grancher, Thaon, and Lépine aided the reaction against the dualism of phthisis. These works, which threw new light on caseous pneumonia, chronic inflammation, and the dissemination of tubercle by the lymphatics, rallied medical opinion in France around the conception of the unity of phthisis. Rindfleisch in Germany, Wilson Fox, Burdon Sanderson in England, gave in their adhesion to the same view; and Charcot gave it the authority of his teaching in the Faculty of Paris.

Among the partisans of the unity of pulmonary phthisis (and we have always been of this number) there are those who, while looking upon the tubercular granulation in its various stages as the initial essential lesion, do not hesitate to give an important place beside it to the various kinds of bronchitis and pneumonia. These latter, though caused by granulations, still possess quite distinct anatomical characters. Such is the general opinion. Others, at the head of whom is Charcot, while referring to tubercle all the lesions observed in the lungs of tubercular subjects, entirely suppress caseous pneumonia and almost entirely all the other pneumonic processes which complicate tuberculosis. We cannot subscribe to this view. Glanders, syphilis, purulent infection, &c., may also produce inflammation and caseous infarctuses in the lungs. It is, moreover, a general law that tubercle always causes inflammation around itself, which varies according to the organ affected, pursues a special course and tends to end in the caseous condition. This special inflammation caused by tubercle should be carefully distinguished from ordinary inflammation, whether called caseous or tubercular inflammation. In anatomical characters it resembles acute or chronic inflammation due to quite another cause than tubercle, which it only resembles by its caseous condition. We discussed the same condition in tubercle of the bones, serous and mucous membranes. We shall describe later the various kinds of chronic pneumonia which accompany tubercle of the lung, and we will give in detail the anatomical characters which differentiate it from tubercle, and in each of the other organs we will show the dissimilarity between tubercular inflammation and granulations. The pulmonary changes in phthisis are very various. We shall first describe discrete and confluent tubercular granulations of the lung, then congestive, catarrhal, fibrinous, and caseous pneumonia, vomicæ, interstitial pneumonia, perforations, bronchitis, dilatation of the bronchi, &c.,

after which we will examine these lesions in groups, and will study the most common anatomical forms of pulmonary tuberculosis.

Tubercular granulations of the pulmonary parenchyma.—We do not intend to redescribe here the tubercular granulation considered generally (*vide* vol. i. p. 199), nor tubercle of the serous membranes (*vide* vol. i. p. 432) and of the mucous membranes (*vide* vol. ii. pp. 53 and 60), but shall proceed to consider the origin and development of the tubercular granulation of the lung. For the moment we shall only consider the granulations of the pulmonary parenchyma, that is to say, of the terminal extremities of the bronchi and of the infundibula, returning later to those of the interlobular septa, of the pleura, and bronchial mucous membrane.

To study tubercular granulations in the lung, a lung should be selected which is scattered over with small miliary nodules hardly visible to the naked eye, and about a month old. These granulations are grey, slightly opaque, hardly perceptible to the sight, hard to the touch, and can be neither isolated nor enucleated. On scraping them with a sharp instrument, or on attempting to dissect or dissociate them with needles, they resist, and fragments of the surrounding tissue are removed with them. The lung containing them is congested, permeable in a great measure to air, tumefied, and red or pink on section. Nuclei of brocho-pneumonia or diffused catarrhal pneumonia are almost constantly seen in the inferior lobes, and particularly on the posterior border; and in these regions the tubercles are located in the midst of an inflamed tissue. On hardening fragments of such a lung in Müller's fluid, gum and alcohol, and staining sections with purpurin or picro-carminate, the tubercular nodule will be seen under a low power to generally consist of a group of pulmonary alveoli filled with small spherical agglomerated elements. These elements, pressed closely together or separated by fibrils, are about the size of normal or atrophied lymph cells, and fill the interior of the alveoli. The border of these cavities is marked by the elastic fibres of the septa, but the normal capillaries on these septa can no longer be recognised. No red blood corpuscles are extravasated either into the alveolus or into the outer alveolar septa. The contents of the alveoli adhere closely to their walls, and the small cells stick close together and form a compact mass, which takes the stain of picro-carminate or purpurin more slowly than the cells of a recent pneumonic exudation. They,

however, stain well if time be given; if, for example, the sections be placed for twenty-four hours in a solution of purpurin, or be immersed for two or three hours in a few drops of picro-carminate in a moist chamber. Under a high power these small cells of tubercle are seen to be sometimes purely granular, sometimes vitreous and semi-transparent. They have generally a nucleus which stains feebly and is more or less atrophied. In a section of a tubercular nodule of this description four, five, or more alveoli may be seen filled with cells. The number of the alveolar cavities depends on the direction of the section. If the section passes through the middle of an infundibulum, a large cavity will be found in the centre of the tubercle into which the peripheral alveoli open; both the central cavity and the alveoli are filled with the same elements. This lesion may exist alone, but the bronchi and blood vessels are often altered at the same time. When the terminal bronchus is divided, it also appears filled with an exudation composed of small agglutinated cells, but at the same time a diffused or nodular thickening may often be observed in the connective tissue which forms its wall as well as in the wall of the final ramifications of the lobular bronchi, which thickening surrounds the bronchial tube for a certain distance. In a transverse section of these bronchi one or more tubercular granulations are then seen either occupying half their circumference or quite surrounding them. These peribronchial nodules, which, according to Rindfleisch and Charcot, are the initial manifestation of pulmonary tuberculosis, are situated, they contend, at the spot where the intralobular bronchi bifurcate. According to Rindfleisch the neoplasm is developed at the spot where the terminal bronchus opens into the alveolar ducts of the acinus; according to Charcot they may be found on larger bronchi. These peribronchial granulations are constituted like all tubercle developed in connective tissue. Their tissue is continuous with that of the neighbouring alveolar septa which is implanted on the fibrous membrane of the bronchus. These septa are themselves thickened (*vide* vol. ii. fig. 37). Similar growths, either small nodules distinctly tubercular in character or diffused neoplasms, are found around the small branches of the pulmonary artery in the neighbourhood of the diseased alveoli. Thus constituted by small coherent cells contained in the alveoli and terminal bronchi, and by a similar infiltration of the peribronchial and perivascular connective tissue, the small pulmonary tubercle measures from $\frac{1}{2}$ to 1 or 2 mm. in diameter. Whatever may be its size, it is

always surrounded from the commencement by alveoli which are all affected either with catarrhal or fibrinous inflammation or by simple congestion. The zone in which the pulmonary tissue is congested and inflamed is more or less extensive. Hence, as will be seen by the short explanation just given, tubercle of the lung is generally complex in character, for the contents of the alveoli and terminal bronchi, the neoplasmic infiltration of the connective tissue, and the walls of the bronchi and blood vessels all simultaneously participate in its formation.

a. **Lesions of the alveoli.**—We have seen that tubercular granulations often originate in the pulmonary alveoli, that is to say, these cavities become filled with cells agglutinated together in the midst of an uniting fibrillar or granular substance. Are the contents of the alveoli sufficiently characteristic to be regarded as a tubercular tissue, or can they be distinguished from the exudation of ordinary pneumonia? Certainly originally the alveolar contents, which soon become an integral part of the tubercle, do not differ from pneumonic exudation. Most neoplasms, whatever they are and however characteristic they become later, commence as a simple inflammation. Often, in fact, the tubercular granulation of the lung seems to have its point of departure in the alveoli, which show at first the lesions of fibrinous pneumonia. The intra-alveolar exudation is composed of fibrin and lymph cells; these cells, present in great numbers, and pressed close together in the midst of a fibrinous mass which becomes granular or vaguely fibrillar, are insufficiently nourished; they then become granular or vitreous, at the same time that the alveolar capillaries are obliterated by the lymph cells stagnating within them. The alveolar contents are henceforth doomed to molecular destruction. But at the commencement they resist; these elements are isolated with difficulty from one another even when it is attempted to separate them in a very fine section. By dissociation the needles only separate blocks of cells agglutinated together. It is most probable that a fibrinous and cellular exudation, similar to that of fibrinous pneumonia, may aid in the development of tubercle, as fibrinous thrombi of the vessels in tuberculosis of the meninges, the peritoneum, and the bucco-pharyngeal mucous membrane become the centre of formation of tubercular granulations of these membranes. In the midst of these alveolar masses of cells a very characteristic giant cell is sometimes found; it is generally situated in contact with the alveolar wall.

Whence come the cells which fill the lumen of the alveoli?

Are they produced by proliferation of the epithelium, or are they only leucocytes extravasated from the blood vessels? Or does thickening of the alveolar wall play some part in obliteration of the alveoli? These three hypotheses may all be maintained, and it is probable that these various modes of cellular formation take part in the constitution of the intra-alveolar tubercular tissue. At the periphery of a pulmonary granulation, a bud may often be seen composed of embryonic tissue, continuous with the tissue of the granulation, and projecting into the lumen of a neighbouring alveolus, which it nearly fills. This bud is con-

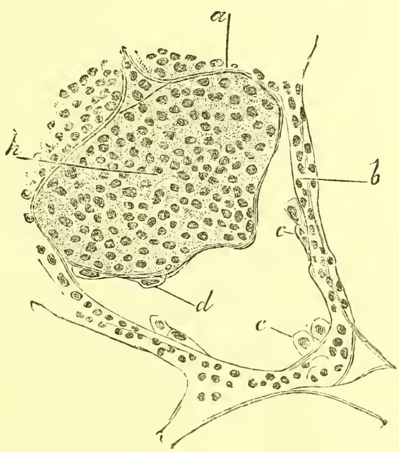


FIG. 63.—SECTION ACROSS AN ALVEOLUS SITUATED AT THE PERIPHERY OF A TUBERCULAR NODULE.

a, limit of the alveolus at the level of the granulation; *n*, tissue of the granulation projecting into the alveolus; *d*, epithelial cells on the surface of this tissue; *b*, septum of the same alveolus lined with epithelial cells, *c*. Magnified 200 diameters.

tinuous at its point of implantation with part of the alveolar wall; its projecting portion is covered by epithelial cells similar to those which line the whole of the alveolar septum.

b. **Lesions of the vessels.**—The capillary vessels and small arteries and veins which are distributed to a pulmonary tubercle are always soon obliterated, that is to say, as soon as the granulation is formed. Injections into the pulmonary artery, and which hence penetrate throughout the lung, as the capillaries of the terminal bronchi communicate with the capillaries of the alveoli, are always arrested at the edge of the tubercles. Hence in sections of a lung affected with acute miliary tubercle, injected with soluble Prussian blue, and then stained with picro-carminate, red patches are observed corresponding to the disseminated

tubercles in the lung the capillaries of which are injected with blue. These preparations are both beautiful and instructive. But before being obliterated by lymph cells and fibrin, the vessels of the inflamed part which eventually becomes tubercular are much dilated and filled with red blood corpuscles. The obliteration of the blood vessels in tubercle is a constant fact. We described in 1867 the fibrinous coagula which are observed in the vessels which pass through tubercular masses of the lung, and which resemble those in the vessels of the pia mater; we described the peculiar endo-arteritis of tuberculosis in serous membranes (*vide* vol. i. p. 433) and in tubercular meningitis (*vide* vol. i. p. 580). The researches made on the origin of the giant cells have led to the discovery that these cells may be developed in the interior of the blood vessels, either in the midst of a

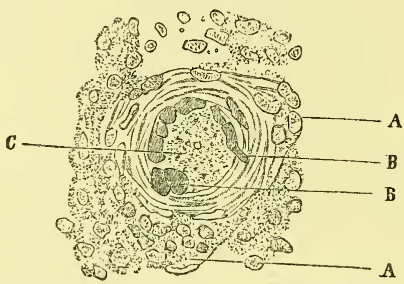


Fig. 64.—Section of a Tubercle of the Brain,

In which the contents of the blood vessel, c, are composed of granular fibrin; a, tubercular tissue; b, white blood corpuscles. Magnified 400 diameters.

thrombus composed of fibrin and lymph cells, or in the inflamed and granulating internal wall. We shall show later that the same process is sometimes seen very distinctly in the mucous membrane. In the same way in the lung, while the tubercle elements infiltrate the peribronchial and perivascular connective tissue and fill groups of pulmonary alveoli, the vessels show the same lesions as the vessels of the serous and mucous membranes. These lesions are not followed so easily in the lung as in the membranes, as the structure of the lung is more complicated; but most of the same facts have been, nevertheless, observed in the pulmonary vessels as in those of the serous and mucous membranes. H. Martin, in his thesis on tubercle (1879), gives a preponderating rôle to the vascular lesions, to inflammation of the tunica interna and externa of the small arteries and veins, and regards them as primary. Endo-arteritis and endo-phlebitis cause growths which

project into the lumen of the vessels and arrest the circulation, producing thromboses in all their divisions as far as the capillaries, the internal coat becoming inflamed in all the vascular branches at the same time. Mügge has observed projecting patches of endo-arteritis and endo-phlebitis, which he considers to be tubercular, in the tunica interna of the large branches of the pulmonary artery and more frequently in the intima of the pulmonary vein. The tunica externa of the same vessels is simultaneously the seat of a more or less considerable infiltration of lymph cells which form a thick ring around them. The peripheral connective tissue is altered in the same way, whence results an accumulation of cells in the form of one or more granulations, according to the size of the vessel. Then at the centre of these perivascular

Fig. 65.—Longitudinal Section of a Lymph Vessel in Tubercular Perivascular Tissue.

b, connective tissue belonging to the external tunic of a pulmonary arteriole; *l*, lymphatic filled with cells; *a*, pigmented connective tissue infiltrated with small cells, and forming part of a tubercle; *v*, vessel full of blood. Magnified 200 diameters.

nodules giant cells are produced, and atrophic granular degeneration of the lymph cells. The lymphatics which participate in the inflammation are easily seen in the external tunic or in the connective tissue at its edge. These lymphatics are filled with round cells, and sometimes with fibrin in a fibrillar condition. Their endothelial cells are sometimes swollen and visible; often, however, the endothelial cells have disappeared, and round cells are alone present in contact with the wall. The lymphatics found on the border of the blood vessels are sometimes large, sometimes small; in fig. 65 one of the latter is represented; their walls blend with the peripheral connective tissue. Morbid connective tissue surrounds these blood vessels, as well as their branches, for a variable distance. This tissue is continuous with the neighbour-

ing alveolar septa, which are thickened and contain a large number of small round cells. The capillaries of the tubercular nodules show similar lesions. They are often dilated at the beginning, and their endothelial cells, larger than normally, are swollen centrally. Presently the lymph cells accumulate within them and the circulation is arrested; coagulated fibrin is then found.

c. **Lesions of the bronchi.**—The lesions of the bronchial tubes in tuberculosis are complicated. We have already pointed out that tubercular nodules are often situated around and occupy the lumen of the terminal bronchi, and that this is, according to Rindfleisch and Charcot, their almost exclusive seat. A terminal or a lobular bronchus is often found at the centre of a section

FIG. 66.—PERIBRONCHIAL TUBERCULAR NODULE.

a, filled pulmonary alveoli; *t*, tubercle; *b*, bronchus. Magnified 80 diameters.

of a tubercular nodule. The bronchus is surrounded by a crescent or ring of embryonic tissue, in which are found one or more tubercular nodules, characterised by caseous degeneration of their centre and by the presence of giant cells. Within the bronchus is an exudation formed of round and free lymph cells. The tunicæ of the bronchus, if it be a lobular or supralobular bronchus, or its single tunica if it be a terminal bronchus, are markedly thickened. The cells of the ciliated cylindrical epithelium are often preserved, but are more or less raised and displaced by the lymph cells which accumulate beneath them and are interposed between them. This lesion of the peribronchial connective tissue, which consists in the presence of a large number of lymph cells situated

between its fibres, has been regarded as simple peribronchitis by Virchow, Colberg, Bouchard, &c. But in places the cells of this tissue are crowded together, undergo atrophy, and become

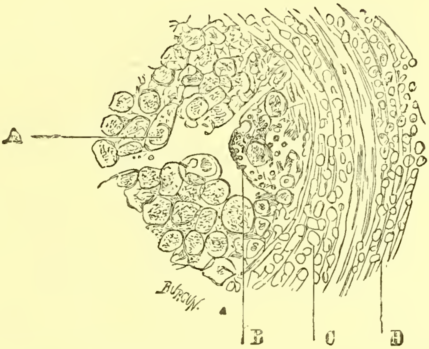


FIG. 67.—SECTION OF A BRONCHUS IN WHICH THE EXUDATION HAS BECOME CASEOUS.

A, agglutinated and granular epithelial and pus cells ; B, fat granules ; C, infiltration of the internal connective-tissue membrane of the bronchus by lymph cells ; D, inflammation of the peripheral cellular membrane of the bronchus. Magnified 200 diameters.

caseous, or a giant cell may be found ; whence results a nodule which partly surrounds a bronchus in a crescentic manner. In other sections many similar nodules are found entirely surround-

FIG. 68.—SECTION OF A BRONCHUS SURROUNDED BY INFLAMED LYMPHATICS IN PULMONARY TUBERCULOSIS.

a, wall ; *c*, epithelium, and *b*, cavity of a bronchus ; *m*, *n*, peribronchial lymph vessels dilated and filled with lymph cells ; *v*, arteriole ; *p*, *p*, pulmonary alveoli. Magnified 20 diameters.

ing the bronchus. Very characteristic granulations are often exactly spherical in shape ; they are always seated in the midst of embryonic tissue which is continuous along the bronchus to its final divisions. The nature of the neoplasm cannot then be

doubted, and we have long considered that this embryonic tissue surrounding the bronchi and blood vessels, and showing from place to place characteristic nodules, is itself tubercular tissue. The muscular tunic of bronchi with three coats is choked by the formation of small cells which infiltrate it as well as the external and internal coats. The internal membrane is often considerably thickened, and it presents buds which project into the lumen of the bronchus, and which, according to Martin, may obstruct it entirely. In this thickened coat the capillaries become first dilated and filled with blood, and are finally obliterated. In other cases, again, the inflamed intralobular and supralobular bronchi continue to secrete a large quantity of pus and to dilate exactly as in capillary bronchitis and broncho-pneumonia. The cylindrical epithelial lining, which is preserved at the commencement of the morbid process, finally falls and is not renewed. The exudation contained in the bronchus dries when the circulation is arrested in the blood vessels; the lymph cells composing it agglutinate together and form a compact and coherent mass, as is represented in figs. 66 and 67. The cells become granular and vitreous, and the contents of the bronchus quite caseous. Around these bronchi lympathics may sometimes be found at the commencement of the morbid process dilated and filled with accumulated lymph cells. These vessels form cellular pools at the side of a bronchus or surrounding it (*vide* fig. 68). Tubercular peribronchitis resembles periarteritis, and is found associated with it. Around the nodules produced, whether in the alveoli or around the bronchi or vessels, a more or less extensive zone of catarrhal or fibrinous pneumonia is always found.

Confluent granulations of the lung.—Around the discrete granulations already described other tubercles are developed, and surrounded, as the first, by catarrhal or fibrinous pneumonia. These unite to form groups; presently these granulations melt together, in consequence of caseous degeneration of the pneumonic exudation contained in the alveoli which separate the tubercles, and a mass about the size of a pea or larger results, formed by the agglomeration of confluent tubercles which are caseous throughout. This caseous degeneration of the primarily inflamed tissue which separates the originally discrete tubercular nodules, and which changes them into a single mass, is found in all tubercular tissues and organs with identical characteristics, for it is always due to the same cause—namely, to arrest of the circulation from

obstruction of the blood vessels. When the circulation is arrested in the blood vessels of tubercles seated in the midst of an inflamed tissue, the majority of, and finally all the blood vessels of the tissue interposed between the tubercles become successively obliterated, and the whole mass, deprived of blood, becomes caseous. One of us has demonstrated this very plainly in the medullary tissue of bone. These confluent tubercles, united by fibrinous or catarrhal pneumonia which has become caseous, form grey or yellowish opaque masses, dry and smooth on section. These patches have often been described as belonging to caseous pneumonia; on examining sections, however, which pass through large tubercles, the primary granulations may be recognised, showing the characters previously described, and in a state of granulo-fatty degeneration more advanced than the pneumonia, equally caseous, which surrounds them. Sometimes these confluent tubercles have undergone fibrous change, described below.

Fibrous granulations of the lung.—Tubercle, as we said when generally describing it (*vide* vol. i. p. 209), shows a great tendency to undergo fibrous change. When tubercle is fully developed, instead of becoming caseous, it often undergoes fibrous change, when it enters upon conditions favourable to recovery, or at least to temporary arrest of the disease. In the lung newly-formed fibres of connective tissue show themselves in the granulations; they are interposed between the cells and predominate more and more; the small cells composing the primary granulation range themselves along bundles of fibres and become fusiform and flattened. Thus at the end of a certain time most of the granulation is composed of fibrous tissue; giant cells are, however, met with either at its centre or periphery. At the same time that the tissue of the granulation becomes fibrous, the septa of the adjacent pulmonary alveoli show a tendency to thicken, and thus interstitial pneumonia is produced around the granulations which have become fibrous. The naked-eye appearance of the pulmonary tissue scattered over with fibroid granulations varies. Sometimes it is but slightly altered, normal or congested, and simply sprinkled over with small semitransparent miliary nodules, which are situated under the visceral pleura as well as in the midst of the lobes. These nodules, which are quite characteristic of tubercle in their naked-eye appearances, are hard to the touch and difficult to separate from the pulmonary tissue, and

still more difficult to dissociate with needles; generally they are neither yellowish nor opaque at their centre.

The fibrous tubercles of the lung result simply from fibrous transformation of the ordinary tubercular nodules; perhaps they may take the fibroid form at once; they are first developed in a group of alveoli, or around the vessels or bronchi; but whatever may be their seat or origin, they develop always in the same manner. In sections of tubercles developed in the alveoli and undergoing fibroid change, the alveolar septa are seen to be thickened, and show a large number of small round cells between the fibrils of the newly-formed connective tissue. These fibrils and trabeculæ of connective tissue are still thicker around the bronchi or blood vessels. The cells inside the alveoli often show then an epitheloid character; the alveolus contracts in consequence of the thickening of its walls, and the epithelial cells are pressed together; a large giant cell, surrounded by epitheloid or round cells, may also be found in some of these contracted alveoli (*vide* fig. 69). Around the bronchi and blood vessels the tubercles are composed of a compact connective tissue formed of fibrils or fibrous fasciculi separated by small round or ovoid cells; these cells have globular nuclei, the fibrous fasciculi stain well with carmine, like the constituent parts of all tissues possessing an active nutritive life. Inside these tubercles giant cells are also found.

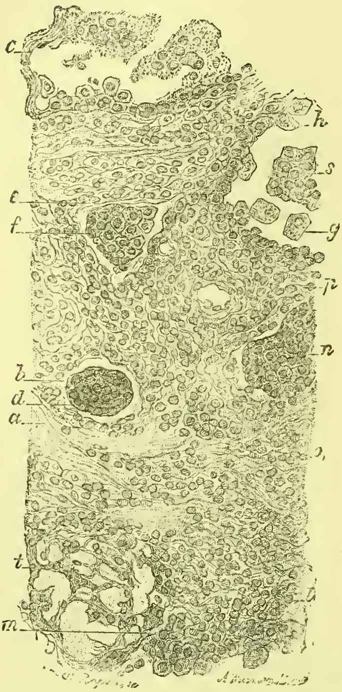


FIG. 69.—SECTION THROUGH A FIBROID TUBERCLE OF THE LUNG.

c, septum of an alveolus situated at the periphery of a tubercle; *h*, epithelial cells attached to the walls of an alveolus; *s*, free agglomerated epithelial cells in the same alveolus; *g*, free isolated epithelial cells; *f*, accumulation of cells in a contracted alveolus, the walls of which, *e*, are thickened; *a*, *p*, *o*, *l*, connective tissue infiltrated with lymph cells, and constituting the tubercular nodule; *n*, *d*, accumulation of cells adhering together in the contracted alveoli. Magnified 150 diameters.

In a more advanced stage of development no indication is given of the primary seat of fibroid tubercles. The nodule, whether it originated in a group of alveoli or around bronchial

tubes or blood vessels, is composed of fibrillar fibrous tissue; the fibrous fasciculi, which are generally thick, often hyaline, and stain pink with picro-carmine, are separated by a large quantity of small round or ovoid cells. Generally the cells at the periphery are rather larger than those at the centre of the nodule. One or more giant cells are almost always found in the small nodule which is quite typical of tubercle, but which also recalls sarcomatous tissue, if sarcoma ever took the form of such small and

Fig. 70.—Section through a Fibroid Tubercle of the Lung.

c, c, c, periphery of the tubercle composed of lymph cells pressed close together; *a, v,* blood vessels containing red blood corpuscles at the periphery of the tubercle; *p,* continuation of the tubercle with the septa of the adjacent alveoli; *g,* giant cell situated near the centre of the tubercle; *d,* small blood vessel full of blood near the centre of the tubercle. The central part of the tubercle is filled with large fasciculi of connective tissue separated by fusiform cells. Magnified 80 diameters.

multiple tumours (*vide* fig. 70). At the periphery of these tubercles their tissue is continuous with the thickened septa of the adjacent alveoli.

In old tubercles, and particularly in fibrous confluent tubercle, a new phenomenon occurs, which might lead to error in an observer not forewarned. The centre of a tubercle or of a collection of tubercles becomes entirely fibrous without the presence of blood

vessels. This fibrous tissue, which contains a very small number of cells, is composed of fine fibrils, close together, staining badly with carmine, and which radiate irregularly from the centre to the circumference of the neoplasm. There are no giant cells in all this central part, which sometimes consists of a rather large patch of confluent fibroid tubercles. At the edge of the patch fibroid tubercles are found similar to those described above and drawn in fig. 70. These have giant cells. Thus, on examining a large

FIG. 71.—SECTION OF A LARGE OLD FIBROUS TUBERCLE SITUATED AROUND A BRONCHUS.

c, part of the circumference of the bronchus; *p*, fibrillar centre of the tubercle; *b*, another fibrous centre of the tubercle; *g*, *g*, *m*, giant cells at the periphery of the tubercle in the midst of a tissue which contains a large number of small cells; *p*, *p*, thickened walls of the alveoli around the tubercle. Magnified 30 diameters.

tubercular nodule under a low power, its centre is seen to contain neither blood vessels nor giant cells, and to stain badly with carmine, while the peripheral zone stains well, and is full of small cells and scattered over with giant cells. This appearance of tubercle under a low power has been well described by Charcot (*vide* fig. 71).

Discrete fibroid tubercle and the periphery of the large confluent

tubercles of the same character show in their interior capillary vessels permeable to blood. These vessels are either newly-formed capillaries or old capillaries which have escaped early obliteration at the commencement of the tubercle. What is certain is that they contain red blood corpuscles which have all the characteristics of these living and circulating elements at the moment of death. The wall of the vessels is very distinct, sometimes rather thicker than in the normal condition, as may be generally observed in sclerosed tissues ; a giant cell often lies quite near to it, without having any direct relation with it. The giant cells in fibroid tubercle are often located in a sac which holds them exactly, composed of encircling fibrous fasciculi ; small round cells are often present between the processes which proceed from the giant cells. The protoplasm of the giant cells is granular or hyaline, stains either yellow or red with picrocarmine ; these processes are continuous with the fibrous fasciculi of the connective tissue, and their nuclei stain a deep red.

Pigmented fibroid granulations.—Old fibroid tubercles frequently undergo black pigmentary infiltration. Some of these tubercles, whether round, small or large, discrete or confluent, are either entirely black or spotted with black. The black granulations fill up the entire tubercular patch, or are only found at its centre or periphery. The black granules, which are composed of particles of carbon or are caused by changes in the blood pigment, are generally found in the cells at their edges, or in the interstices of the fibres. Their abundance depends on the number of cells, and this explains their unequal distribution in a tubercle; if, in fact, the centre of the latter is entirely fibrous without cells, these being only found at the periphery, pigmentation will chiefly be found in the peripheral zone. In the middle of pigmented tubercles, or in their external zone, giant cells are sometimes met with infiltrated with black pigment ; they may attain a great size, and the black granules are generally deposited in the protoplasm around the nuclei. Pigmented fibroid granulations are either entirely or partly non-vascular ; near them the alveolar septa are thickened, and all the lesions of slate-coloured interstitial pneumonia are found. At other times a small number of pigmented granulations are found ; these are the vestiges of a very localised and important tuberculisation which has been extinguished if not cured. The fibrous tissue and giant cells of these tubercles seem to be tolerated, without causing trouble. This is a form of tuberculosis which may be looked upon as curable or as cured ;

it is discovered sometimes at the post-mortem examination of a person accidentally killed by some other cause. Lungs, which contain a large number of fibroid tubercles of more or less old standing, are often attacked, at the end of life in phthisical subjects, by catarrhal or fibrinous pneumonia around the fibroid granulations; they show the peculiar lesions described later on.

Tubercular granulations of the interlobular septa and of the interlobar pleura.—Sometimes at the autopsy of phthisical patients semitransparent granulations are found, fully developed or fibroid, seated chiefly on the surface of the lung, either in the visceral pleura or in the pulmonary tissue. On the lung some interlobular septa are seen to be thickened, fibroid, slightly transparent, and scattered over with tubercular granulations. The pleura which lines the contiguous surfaces of the pulmonary lobes is thick and changed into a single fibrous membrane which solidly unites the two lobes. This membrane is itself scattered over with granulations. Instead of the adhesion of the folds of pleura being complete, short filamentous or membranous fibrous bands are found, and in these granulations are developed either in their substance or on their surface. In sections of the interlobular septa typical granulations are seen under the microscope, and the lesions of the lymphatics, which are numerous, are easily studied in these septa.

Lesions of the lung which accompany tubercle.—We have already seen that recent or fully developed tubercles, whether discrete or confluent, are surrounded by a zone of pulmonary congestion, and of catarrhal or fibrinous pneumonia. Later, when they become fibroid, they show a tendency to induce interstitial pneumonia around themselves, which does not, however, prevent the other forms of pneumonia being present. These inflammatory lesions are more or less extensive. It is invariably the rule that every granulation or every collection of tubercles is surrounded by a zone of inflamed pulmonary tissue; but in the ripe or fibrous condition of the tubercles the pneumonia, whether catarrhal or fibrinous, often invades lobules, groups of lobules, a large part of or an entire lobe. The portions of the lung affected by pneumonia contain a various number of caseous or fibrous tubercles, but, on the other hand, no tubercles may be present for a rather large extent of the inflamed pulmonary parenchyma. Thus pneumonia in all its forms plays an important part in phthisis,

and most of the lesions observed in the lungs of phthisical subjects are due to its active influence.

a. **Pulmonary congestion.**—The portions of congested lung which surround tubercles show the lesions already described on p. 84: namely, distension of the blood vessels of the alveolar septa by red blood corpuscles, tumefaction of the epithelial cells, effusion into the alveoli of a fluid which sometimes contains fibrils of fibrin, red blood corpuscles, leucocytes and epithelial cells detached from the wall. The gross appearances of congestion are a red colour, and tumefaction of the lung which is more or less infiltrated with a red or pink spumous fluid; the patch of congestion varies in extent. In certain cases of acute granular phthisis congestion constitutes together with bronchitis and tubercular granulations the predominating lesion.

b. **Catarrhal pneumonia.**—Instead of being surrounded by a tissue which is simply congested the tubercular granulations are often located in the midst of alveoli which show all the lesions of catarrhal pneumonia: namely, distension of the blood vessels, and desquamation of the epithelial cells, which are round or polyhedric

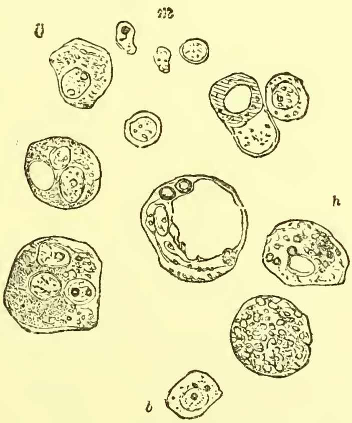


FIG. 72.—ELEMENTS IN A STATE OF GRANULAR DEGENERATION, FROM A CASE OF PNEUMONIA UNDERGOING RESOLUTION.

a, granular body; *b*, *b*, pavement cells; *h*, vesicular cells and white blood corpuscles; *f*, a pavement cell containing two nuclei and an empty space; *m*, granular fragments. Magnified 500 diameters.

in form, from the alveolar walls, and the alveolar cavities are filled with a large number of lymph cells and a few red blood corpuscles. These cells are more or less charged with fat granules, and often in the midst of the intra-alveolar exudation or against the alveolar wall large spherical cells are found which contain three,

four, or more nuclei. In the protoplasm of some cells vacuoles are seen with serous or colloid contents; sometimes also in the alveolus large cells are observed which have undergone vitreous change throughout their whole mass; these stain orange yellow with picrocarminate. The intra-alveolar cells are spherical, prismatic, and irregularly polyhedric with blunt angles; they are often the only formed elements observed, the exudation containing very

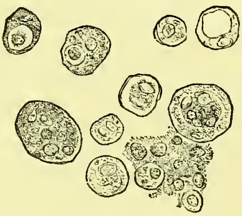


Fig. 73.—Isolated Elements from an Exudation in Tubercular Broncho-Pneumonia of recent date. Magnified 200 diameters.

few or no lymph cells. This zone of catarrhal pneumonia is thinner around recent granulations, and it is continuous at its periphery with the congested pulmonary tissue. Many adjacent granulations surrounded thus with catarrhal pneumonia constitute an irregularly spherical nucleus, which may extend to one or more lobules; it is seated in the midst of a congested lung. One or more lobules of catarrhal pneumonia may be found in a phthisical lung in the midst of inflamed tissue without tubercular granulations being present. Further, at the base of the superior lobe and at the base of the posterior border of the inferior lobe, rather extensive and diffused catarrhal pneumonia may be not unfrequently found which has invaded half, or even more, of an entire lobe, the inferior lobe in particular. In the hepatised part disseminated tubercles are generally observed, but sometimes very few are present, so that there does not seem to be here the relation of cause and effect between tubercles and pneumonia; in fact, the inferior lobes, which are most generally affected by diffused pneumonia, are more rarely and less abundantly invaded by tubercles than the superior lobes. Surprise must not therefore be felt at seeing the pneumonic process in tuberculosis apparently independent up to a certain point of tubercles, and developed parallel to them without absolutely following their distribution. Catarrhal pneumonia is, as we have already shown (*vide* vol. ii. p. 91), chiefly dependent on bronchitis. In all these cases of tuberculosis acute bronchitis is present and

is propagated to the terminal bronchi; it is often accompanied with tubercles of the mucous membrane of the medium-sized and large bronchi and trachea, and is characterised by the presence of numerous lymph cells in all the layers of the mucous membrane. As in all acute capillary bronchitis, the infundibula and alveoli are inflamed and affected with catarrhal pneumonia; and, as in simple broncho-pneumonia, the bronchi are generally dilated. This catarrhal pneumonia, which may be often studied at its commencement at autopsies of persons who have died of rapid or subacute phthisis, shows, on microscopical examination, the same anatomical lesions as simple catarrhal pneumonia;—namely, engorgement or splenitisation at its commencement; later, soft hepatisation, pinkish grey or grey in colour, finely granular and yielding on scraping a greyish fluid more or less mixed with blood. What is peculiar in this pneumonia of phthisis is that it does not undergo resolution and often passes into the caseous condition. For a varying extent around the tubercular granulations the hepatised pulmonary tissue becomes grey, dry, compact, finely granular, and yields no fluid on scraping. Lobules of caseous pneumonia, in every way similar, also exist without tubercular granulations being present inside them. On examining sections of this hepatised tissue under a low power the delicate alveolar septa are seen perfectly distinctly; they are well characterised by their elastic fibres, and they limit alveoli or infundibula which are filled by a slightly transparent and granular exudation. The blood vessels of the alveolar septa are no longer visible, for they are empty of blood and have collapsed or become obstructed with fibrin; the arterioles are equally obliterated. If the cellular elements contained in the alveoli be examined either in sections or after dissociation, it will be seen that they consist of small lymph cells, vitreous or slightly granular in appearance, irregular by reciprocal pressure, and containing fine proteic or fatty granules; at the same time polygonal or round cells are found, varying in size, uni- or multi-nucleated, and in which both protoplasm and nuclei have undergone granular degeneration; the nuclei are not always visible, and these elements have lost cellular life. They also divide into small angular fragments, which Lebert called tubercular corpuscles and regarded as characteristic of tubercle. The intralobular bronchi distributed to these lobules or nuclei of catarrhal pneumonia undergo lesions similar to those of the infundibula and alveoli. The bronchial cavity, deprived of its ciliated cylindrical epithelium, contains either living lymph cells or lymph

and epithelial cells agglutinated together and in a condition of vitreous or granular degeneration. These cells then constitute a

Fig. 74.—Section of a Lung affected with Lobular Caseous Pneumonia.

a, pulmonary alveoli filled with inflammatory exudation ; *b*, infundibulum. Magnified 40 diameters.

caseous mass often adherent to the bronchial wall. It need not

Fig. 75.—Elements in a state of Granular Degeneration, from a case of Pneumonia undergoing resolution.

a, granular body ; *b*, *b*, pavement cells ; *c*, vesicular cells and white blood corpuscles ; *f*, a pavement cell containing two nuclei and an empty space ; *m*, granular fragments. Magnified 500 diameters.

be repeated that the intralobular bronchi may present peribronchial tubercles similar to those drawn in fig. 37.

The nuclei of catarrhal broncho-pneumonia just described may, whether in the caseous condition or not, or containing more or less tubercular granulations, be invaded throughout their whole extent by destructive suppuration, and cavities may be produced. Every observation tends to the belief that suppuration and destruction of the lobule is brought about by the mechanism described above when treating of the peribronchitis of non-tubercular catarrhal pneumonia (*vide* p. 95). The terminal and intralobular bronchi are acutely inflamed and filled with lymph cells; the bronchial wall is infiltrated with the same elements; its connective-tissue fibres, compressed by the lymph cells, are destroyed and replaced by pus. The alveoli adjacent to the bronchus, and the infundibula and alveoli dependent on it, are filled with pus and open into a purulent focus which replaces the terminal bronchus; their septa are gradually destroyed, and a small vomica is produced, which increases from the centre towards the periphery by ulceration of the hepatised nucleus. A zone of this hepatised tissue persists for a long time at the periphery of the cavity, but it is slowly eliminated and finally disappears. The cavity is thus bordered by an inflamed, vascular, and living tissue, covered with fleshy granulations, such as are observed at the borders of all ulcers. The tubercular granulations which existed in the hepatised patch are destroyed and eliminated as the tissue is affected by pneumonia. Cavities caused by caseous broncho-pneumonia are produced by another process. A lobule, formed of a tissue in which tubercles and pneumonia in the caseous condition, obliterated blood vessels, and small bronchi filled with a caseous exudation simultaneously exist, is present in the lung like a foreign body; suppurative inflammation cannot be developed at its centre, but in the surrounding pulmonary tissue and in the bronchus at the point where obliteration occurs suppurative inflammation is set up and gradually eliminates the whole of the softened and broken-down caseous nodule.

This form of caseous broncho-pneumonia with peribronchitis, dilatation of the bronchi, and formation of small cavities, is that which is most frequently found at the autopsy of patients who have succumbed rather rapidly, that is to say, a few months or a year after the commencement of tuberculosis. The formation of vomicæ is so rapid, when the lobules destroyed by suppuration are seated immediately under the pleura, that perforation of this membrane is sometimes produced. The delicate and friable wall formed by the inflamed pleura, which is infiltrated with pus cells

and covered with fibrinous false membranes, becomes ruptured and pneumo-thorax is produced. Such a perforation may be, however, temporarily plugged by the fibrinous false membranes of the pleura. As the result of the pneumo-thorax and purulent pleurisy which then occur the lung collapses completely, unless it is surrounded by a resistant fibrous pleural sac. In such a case multiple lesions are observed, discrete or confluent tubercles, nuclei of pneumonia in various stages, vomicæ, &c., and the whole is surrounded by atelectasic pulmonary tissue.

c. **Fibrinous pneumonia.**—Fibrinous pneumonia, associated with pulmonary tubercle, is at least as common as catarrhal pneumonia. It is characterised by the presence of an exudation in the alveoli which is rich in fibrillar fibrin, and entangled in the large meshes, which are composed of thick and numerous fibrils, are lymph cells as characteristic as those of acute lobar pneumonia. The progress of this lesion in the lung and its relations with tubercular granulations are the same as in catarrhal pneumonia. Thus around discrete or confluent tubercles a more or less extensive zone may be observed, in which the alveoli and small bronchi are filled with a fibrinous exudation; beyond this zone the pulmonary tissue is simply congested, or, as may occur sometimes, it is affected with catarrhal pneumonia. Thus patches of fibrinous pneumonia affecting a small group of alveoli are sometimes seen as large as a millet or mustard seed around a granulation; sometimes larger, the size of a lobule or more, when many neighbouring granulations or confluent tubercles are surrounded. Diffused masses of fibrinous pneumonia, the distribution of which resembles that of catarrhal pneumonia, are rather frequently found disseminated in the inferior lobes, the middle lobe, or at the base of the superior lobe. As in the latter lesion, it is not unusual to find homogeneous masses of fibrinous pneumonia in the midst of which tubercles are very rare or even altogether absent.

The lesions observed under the microscope of this form of fibrinous pneumonia differ in a marked manner from those which characterise acute pneumonia; its slow development and its termination by caseous degeneration differentiate it entirely. The parts affected with fibrinous pneumonia rarely present to the naked eye an appearance of engorgement and red hepatisation; at the edges, however, of the parts which are in a condition of grey hepatisation pulmonary engorgement or red hepatisation may sometimes be found; the spots are, however, always much smaller than the granulations of acute idiopathic pneumonia. Sometimes

recent fibrinous pneumonia, extending to a portion of a lobe or to an entire lobe, the inferior, for example, may be observed at the post-mortem examination of patients who have died suddenly in the course of acute phthisis or at the end of chronic tuberculosis. The surface of the divided hepatised lobe is then red or grey, finely granular, and more or less gorged with blood; on scraping a thick grey or red fluid is obtained containing grumous particles yielded by the intra-alveolar fibrinous exudation. These appearances closely resemble those of acute idiopathic fibrinous pneumonia, or of fibrinous broncho-pneumonia with disseminated nodules. Though in the early stages this pneumonia differs but slightly from acute pneumonia, this is no longer the case when more advanced. Generally the hepatised parts are grey, dense, homogeneous, and are of a certain hardness; on section they show a grey, plain, non-vascular, dry surface; they yield no blood-stained fluid on scraping, but simply a little transparent serum. What particularly characterises this grey hepatisation is its semitransparency, and a dull *reflet* its surface gives on section. Its tissue is easily dilacerated, but the morsels into which it is separated preserve their form. In the midst of this quite peculiar hepatisation the opaque tubercular granulations, yellow centrally or throughout, are easily seen, whether isolated or confluent, as the hepatised lung is slightly transparent. Tubercular granulations may, as we have already said, be absent in some of these patches.

In the early stages of engorgement and red hepatisation the microscopical lesions do not differ from those of acute pneumonia, or rather of pseudo-lobar broncho-pneumonia; but this is no longer the case in the stage of grey hepatisation just described. The lesions are entirely distinct from those of a simple acute pneumonia. In sections examined under a low power it will be seen that all the alveolar and bronchial cavities are filled with exudation, and that the septa are not thickened except in the neighbourhood of tubercles developed around the bronchi or blood vessels. We know that in acute pneumonia the fibrin breaks down at the end of the red hepatisation stage, and that in this period, as well as in that of grey hepatisation, there are no tumefied epithelial cells along the alveolar wall, the whole alveolus being completely filled with lymph cells. When resolution occurs the epithelial cells reappear on the walls, but the fibrin has then completely disappeared. In the fibrinous pneumonia which accompanies tubercle a layer of large cells are, on

the contrary, generally found lining all the alveolar septa. These cells are large, irregular in form, polyhedric, with swollen protoplasm, and contain one ovoid nucleus. They stain well with carmine and purpurin. Some contain two, three, or even more nuclei (*vide* fig. 76). Inside this layer there are a few free

FIG. 76. SECTION THROUGH A LUNG AFFECTED WITH FIBRINOUS PNEUMONIA IN TUBERCULOSIS.

p, wall of the alveolus; *d*, its capillary vessels filled with red blood corpuscles; *a*, lymph cells; *b*, *c*, large tumefied epithelial cells attached to the wall; *m*, granular lymph cells contained in the network of fibrils of fibrin *n* and occupying the central cavity of the alveolus. Magnified 300 diameters.

lymph cells, then a tangled mass of fibrils of fibrin and lymph or epithelial cells, which fills up the rest of the alveolar cavity. The fibrils are rather thick and their edges clearly defined; they form large meshes within which are found lymph cells and detached epithelial cells; they stain pink with picrocarminate. The cells contained in this fibrillar network are sometimes not very numerous; it is very rare to find red blood corpuscles among them. Exactly the same lesions are found in the small bronchi. The fibrillar fibrin enclosing round cells in its meshes penetrates from the small bronchi into the infundibula and thence finds its way into the alveoli. At this stage the alveolar capillaries are permeable to blood; sometimes they are even dilated. There is no doubt that the fibrinous exudation may remain in this condition for a considerable time; how long we cannot positively say, but certainly much longer than the duration of fibrinous hepatisation in acute pneumonia. Later this grey hepatisation passes into the caseous condition and is recognised by its grey or yellow colour and by a peculiar opacity and dryness. In the parts which have undergone caseous change the alveoli are completely filled

with exudation, the septa are not generally thickened, and their capillaries are impermeable to blood. On studying under a high power sections of the lung thus altered the large epithelial cells of the wall will be seen to have disappeared, and each alveolus is filled with filaments of fibrin, between which lymph cells are accumulated; they are granular, atrophied, or slightly transparent; their nuclei are less visible and stain indifferently with carmine. When these elements are pressed closely together, the fibrin which separates them is seen with difficulty; it is less distinctly fibrillar in appearance, the fibrils are irregular and shorter, and it often becomes rough or homogeneous; but all the intermediate states between fibrillar and amorphous fibrinous exudation may be followed and recognised.

Fibrinous pneumonia in the caseous state corresponds exactly in its macroscopic characters to grey semitransparent infiltration and to the yellow infiltration of Laennec. It may, we repeat, invade a greater or less area of the lung, either by isolated nodules or by extensive diffused masses. Most frequently quite distinct tubercles are observed in the centre of the nodules, recognisable by the naked eye as well as by the microscope, but sometimes these pneumonic masses show very few tubercles or none at all. This form of pneumonia is sometimes uniformly developed throughout a lobe, or more rarely extends to a large portion of one of the lungs; it is then known under the name of caseous lobar pneumonia. Isolated or confluent tubercles are generally met with in the caseous state, but they are also sometimes fibrous. We have observed fibroid tubercles, evidently of old date, occupying the centre of fibrinous pneumonic patches, either lobular or pseudo-lobar, of several weeks' duration, and we have been able to clinically follow their rapid progress. Fibrinous lobar or lobular pneumonia is always accompanied by fibrinous pleurisy or proliferating pleurisy, when it is extensive and when the diseased lobules reach the surface of the lung.

The destruction of nuclei of fibrinous caseous pneumonia and the formation of cavities take place by the same mechanism, which we have before described when speaking of catarrhal pneumonia; that is to say, by peribronchial inflammation and by suppurative eliminative pneumonia, which dissociates the mortified and disintegrated parts, and carries them away in the sputa. The cavities directly communicate from the outset with the inflamed and often dilated bronchi. In more extensive patches of caseous fibrinous pneumonia fissures are sometimes seen in the

midst of the gangrenous mass; they are produced by the molecular disintegration of the tissues. When these fissures open a bronchus permeable to the air, it becomes inflamed and secretes a large quantity of pus, which aids the softening and expulsion of the mortified parts. The pus contained in the bronchus and the adjacent inflamed pulmonary tissue is mixed with a caseous detritus contained in the cavity which is being formed. The walls of the cavity are anfractuous, and more or less voluminous fragments of the hepatised and caseous lung are partly detached and suspended from them, and the whole undergoes progressive destruction. It is thus that great cavities are formed in the midst of lobar or pseudo-lobar tubercular pneumonia.

Cavities.—We have just seen that cavities are formed when suppurative and eliminative broncho-pneumonia occurs in a lobule, or in a large patch of catarrhal or recent caseous fibrinous pneumonia; this gangrenous patch almost always contains tubercular granulations in a state of caseous degeneration. In a cavity which is still extending we find in the tissue which is not yet destroyed all the lesions of caseous pneumonia, whether this tissue forms an adherent layer or is already partly detached and floating in the cavity. The inflamed and highly vascular peripheral pulmonary tissue forms a layer of embryonic or fibrous tissue. If the cavity occupies the place of a diseased lobule, the loss of substance is limited exactly by the perilobular fibrous tissue. Whatever be the mode of formation and the volume of the cavity, it always communicates from the outset with a bronchus, which is often dilated. The internal coat of these bronchi is always highly inflamed, and stops suddenly at the ulcerated edge of the loss of substance. Many small cavities may communicate with one another, owing to the suppurative destruction of the hepatised tissue which separated them. When of recent formation the walls of the cavities are covered with thick, creamy pus; later the internal wall, at first infiltrated with pus and pulpy, becomes lined externally by a dense fibrous tissue, which is formed under it, and by interstitial pneumonia, which establishes a passage between the wall of the cavity and the normal or slightly altered pulmonary tissue which surrounds it.

On making sections of the wall and adjacent pulmonary tissue of a cavity of two to three months old, and on staining them with picrocarminate, the following arrangement is generally found: On the surface of the loss of substance pus cells are found either free or united into a more or less thick layer; beneath these ele-

ments is a layer formed of very vascular embryonic tissue, containing large blood vessels with a single coat. These vessels are filled with red blood corpuscles; some of them, however, are accidentally obliterated by a clot containing lymph cells and fibrin. This zone of embryonic tissue is limited by an internal surface, which is sometimes smooth and regular, sometimes granulating. Epithelial cells are never found on this free surface. Beneath it is found a layer of fibrous tissue, the fibres of which are generally parallel to the surface of the cavity. In this layer, which is almost always pigmented, a few perfectly characteristic tubercular granulations are generally met with, accompanied by giant cells, at the level of which the blood vessels are obliterated. From this zone of fibrous tissue or interstitial pneumonia, which constitutes the solid framework of the cavity, thickened alveolar trabeculæ spring, so that between the fibrous layer and the pulmonary tissue a zone of interstitial pneumonia may be seen. This is characterised by the narrowing of the alveoli; and they are bounded by thick pigmented fibrous septa, containing large cells filled with yellow or black pigment.

The internal surface of old and extensive cavities is often covered by grey caseous pus; the embryonic layer around it is pulpy and grey, and resembles a low form of granulating tissue. Sometimes the surface of the cavity is dry and smooth, and of a slate-coloured grey. In the first case the free pus cells and those which infiltrate the embryonic tissue are granular and caseous and contain fat granules; in the second case the layer of embryonic tissue is very thin and contains few pus cells. Even when the vomicæ secrete a considerable quantity of pus they are never entirely filled with it, for they always contain a considerable quantity of air. On the surface of old cavities slightly prominent tubercles are often found; they belong to the fibrous coat of the wall. Sometimes the fibrous tissue which constitutes the internal surface, and even the entire wall, of old cavities is dense and corrugated. Very often thick cylindrical trabeculæ project from the wall of the cavity and are thrown like bridges across its interior. These trabeculæ are rather long, round, and about the size of a goose quill; they are generally of a slate-grey colour, or like the surface of the cavities through which they pass. At first sight one is inclined to think that they are bronchi or large vessels, which have resisted destruction of the pulmonary tissue; on hardening them, however, and making transverse sections, it will be seen that they are almost always developed on an uniform

model, and that they very rarely contain vessels more than half a millimetre in diameter. In section they will be seen to be composed of two zones—1st, a rather thick peripheral zone formed of embryonic tissue, pierced by blood vessels and identical in structure with the internal coat of the cavity; 2ndly, a central zone, consisting of a pigmented fibrous tissue sprinkled over with more or less numerous caseous tubercular granulations and giant cells. This tubercular tissue of the central part of the trabeculæ is but slightly vascular; very rarely blood vessels and small bronchi are observed in them.

Aneurisms with soft walls are sometimes seen on the surface of large cavities; these may rupture and give origin to rapidly fatal hæmoptysis. These aneurisms are sometimes rather difficult to recognise, for their soft and greyish wall, often torn and collapsed, resembles a layer of fibrin and is not always very evident, particularly after the surface of the cavity has been washed by a stream of water. But on removing with care the blood extravasated on the surface of the cavity, a small projection may be discovered in which a rent is found plugged by coagulated blood. The aneurism represented below at fig. 77 is very characteristic,

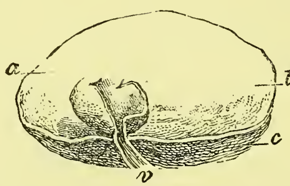


FIG. 77 —ANEURISM DEVELOPED ON THE INTERNAL SURFACE OF A CAVITY.

a, sac of the aneurism, a part of which has been removed, and the vessel, *v*, opened which communicates with it; *b*, the internal surface of the cavity; *c*, a section of its wall.

for the vessel is seen with which it communicates. These aneurisms are developed on the small arteries, which ramify below or within the embryonic layer of the cavity. The wall of one of these small arteries inflames at a certain point, its elastic coat is destroyed, its internal and external coats are changed into embryonic tissue, and the artery dilates by a process already described (vol. i. p. 505). An artery need not be large to become the starting-point of a rather large aneurism; fig. 78 shows a very small artery, *b*, *b*, which opens directly into an aneurism relatively large. In this drawing it will be seen that the elastic coat has disappeared in the wall of the aneurism. The wall is composed of a very thick internal membrane formed of layers sepa-

rated by flat cells, and of an external coat also much thickened, and which is directly continuous with the wall of the cavity. These aneurisms were described by Rasmüssen. We refer their formation to a mechanism which always produces vascular dila-

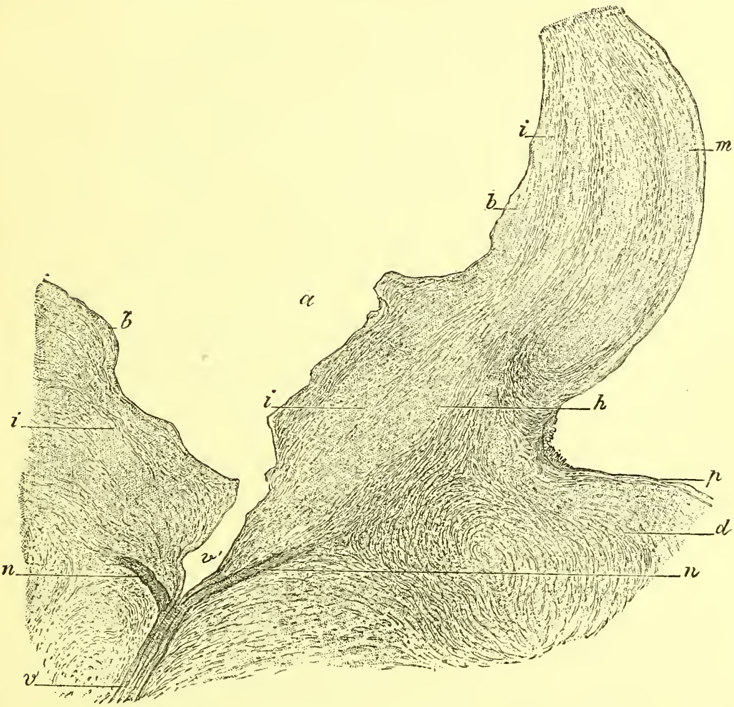


FIG. 78.—SECTION OF AN ANEURISM IN THE WALL OF A CAVITY AT THE POINT WHERE IT COMMUNICATES WITH THE ARTERY WHICH SUPPLIES IT.

b, small artery which opens into aneurism *a* at *v'*; *n*, *n*, middle coat of artery; *i*, *i*, internal coat; *b*, *b*, internal wall of the aneurism; *h*, limit of the internal and middle coats; *m*, wall of the aneurism representing the much thickened external coat of the artery; *p*, wall of the cavity; *d*, connective tissue of the cavity. Magnified 20 diameters.

tations. They have been studied also by Debove, and quite lately Meyer has given a description of them based on many observations. This anatomist, adopting the opinion of Recklinghausen, has insisted on the presence in the aneurismal wall of a hyaline substance, which is present in large quantities; but its *rôle* and chemical nature are not yet well defined.

Old cavities in which inflammation is extant, and which may be considered as almost cured, show a soft internal surface which looks as if it were covered with mucous membrane; no epithelium, however, is present, but simply a layer of round or cubic epithelial cells. These cavities are sometimes continuous with a

dilated bronchus, and they may often be mistaken for the terminal dilatation of a bronchus. We have already said (vol. ii. p. 66) that tuberculosis is the most common cause of bronchial dilatations. The cavity in which the surface is smooth may suppurate anew, and part or its whole surface may ulcerate. The fibrous tissue which lines external cavities may, when located near the surface of the lung, be continued to the visceral pleura, which then becomes thickened, fibrous, and adherent to the costal pleura. In this case subclavicular depression is caused when, as is usually the case, the cavities are situated in the apex of the lung. Communications may be established between such cavities and a lymphatic caseous focus at the root of the bronchi or an abscess caused by Pott's disease, and even with the external air by means of a fistula.

Interstitial pneumonia.—We have already said (*vide* p. 146) that fibrous and pigmented tubercles generally cause thickening of the septa of the lung around themselves, and that they are often located in the midst of indurated connective tissue in which the alveolar cavities are no longer visible. This tissue is pigmented, grey or black, and the tubercular granulations which it contains show those changes of colour in their small and large cells, which have been already described. Around these masses, which present no signs of pulmonic structure, the pulmonary tissue shows all the lesions of interstitial pneumonia. When the whole apex of a lung is thus riddled by large old cavities surrounded with indurated tissue, the upper part of the superior lobe, or the entire lobe, is changed into callous tissue, more or less pigmented and slate-coloured. Patches of tubercle in different conditions or nodules of caseous pneumonia of catarrhal or fibrinous origin are often found at the same time. The lesions that we have described, though considered separately, are in fact generally grouped and found in the same lung in different stages. There is, however, a form of tuberculosis in which the granulations, most frequently fibroid and distributed in patches, are accompanied with pigmented interstitial pneumonia. In sections of such a lung examined by the naked eye hard nodules are found, varying in size from that of a pea to a nut and spherical or irregular in outline. These nodules contain tubercles which are either semitransparent or opaque centrally, and are continuous at their periphery with a hard grey or black pulmonary tissue. These patches cannot be torn or indented with the finger nail; they are disseminated everywhere in the lung, but their most usual seat is in the middle of

the lungs, the apices of which are filled with cavities surrounded with interstitial pneumonia, the bases being scattered over with a few discrete tubercles. This predominance of black or grey interstitial pneumonia, forming nodules in the midst of which numerous granulations are found, does not prevent lobules or patches of caseous pneumonia existing in the same lung. These patches of interstitial pneumonia and fibroid granulations may also have small cavities formed in their centre, caused by suppurative bronchitis or by peribronchitis. Examples of this kind may be daily observed; they have been well described by M. Thaon in his thesis.[1] In these cases tuberculosis pursues a rapid course. Whatever may be the seat or mode of origin of pigmented interstitial pneumonia, it always shows the following histological lesions: thickening of the septa by means of connective-tissue fibres and flat cells, the protoplasm of which contains black pigment; incomplete filling of the alveoli by large round black

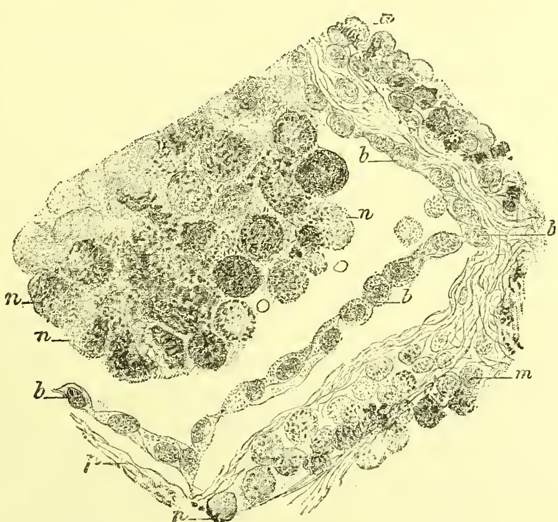


FIG. 79.—SECTION OF A LUNG AFFECTED WITH INTERSTITIAL PNEUMONIA, WITH PIGMENTATION OF THE CELLS.

a, *m*, pigmented lymph cells located in the alveolar septa; *b*, *b*, *b*, tumefied epithelial cells forming a delicate membrane on the surface of the alveolar wall, from which it is partly detached; *n*, *n*, *n*, round pigmented cells in the centre of the alveolar cavity. Magnified 300 diameters.

cells; lining of the alveolar septa by a layer of large tumefied epithelial cells, which are swollen centrally and have large nuclei (these form a kind of membrane); narrowing of the alveoli, &c.

[1] *Recherches sur l'Anatomie Pathologique de la Tuberculose*, 1873.

Emphysema of the lung in phthisis.—Pulmonary emphysema is rather common in the lungs of phthisical subjects. Two forms of it are seen, common emphysema and that variety which is caused by interstitial pneumonia; these have already been described.

Lesions of the bronchi in phthisis.—Lesions of the bronchi play a very important part in the course of phthisis. These organs are always the seat of congestion and catarrhal inflammation in every case of commencing phthisis, at the time that the tubercular granulations are being formed. These may be seated in the submucous tissue or in the peribronchial connective tissue. The bronchitis, which is continuous throughout the whole duration of the disease, accompanies catarrhal and fibrinous, lobular and pseudo-lobar pneumonia, and plays an important part in the formation of cavities (*vide* p. 156). The bronchi are, moreover, often dilated in phthisis, as the result of changes already described in simple broncho-pneumonia. Finally, when the cavities opening one into another communicate widely with the final divisions of a dilated bronchus, and when these cavities themselves have a smooth and dry internal surface, much hesitation is often experienced in deciding the diagnosis between simple bronchial dilatation or dilated bronchi opening into cavities. But the nature of the cellular lining of these cavities, added to the presence of tubercle in their walls and the state of the entire lung, enables one to solve the question.

Anatomical and clinical forms of pulmonary tuberculosis.—In our anatomical study of phthisis we have hitherto analysed in detail and considered separately each pulmonary lesion; it remains now to examine the general distribution of these lesions and the gross appearances of a tubercular lung considered as a whole. The lungs of the same subject may show all lesions from recent granulations to fibroid tubercles and cavities, and from engorgement to caseous or interstitial pneumonia. But these lesions are nevertheless generally developed in a certain order according as the case is one of acute, subacute, or chronic phthisis.

a. **Acute phthisis, generalised granular pulmonary phthisis.**—In this form of phthisis the two lungs are riddled by a considerable number of recently formed tubercles. Other organs, the peritoneum, the kidneys, the liver, &c., often show a more or less confluent eruption of similar granulations; but the tubercles

are sometimes limited entirely to the lungs, bronchi, trachea, bronchial glands, and pleura, that is to say, to the entire respiratory system. Sometimes these granulations constitute the sole pulmonary lesion, but they are often accompanied with catarrhal or fibrinous pneumonia.

In the first case (**acute granular phthisis**) the lungs are swollen, tense, and of a normal or pink colour. On the visceral surface of the pleura very evident and numerous granulations are found developed in the serous membrane itself and in the superficial layers of the lung. They are also found on the parietal pleura, which is often covered with thin fibrinous false membranes. On dividing the lungs, numerous nodules are everywhere seen, regularly disseminated over the grey, pink, or red basis of pulmonary parenchyma; these nodules are composed of tubercles which are semitransparent or opaque at their centre. Though they appear to the naked eye to be quite free of any pneumonic complication, it may be ascertained by microscopical examination that they are surrounded by a zone of alveoli which show all the lesions of catarrhal pneumonia, fibrinous pneumonia, or congestion. Moreover, on studying these granulations more carefully they are found to be not all of the same age. Generally they are found grouped at the apex of one of the lungs or both lungs; they may be caseous, and minute cavities are often found either of recent formation or antecedent to the eruption of generalised tubercle which characterises acute phthisis. On the other hand the granulations of the inferior lobes are quite recent, smaller and more disseminated than those of the superior lobes. In a word granular phthisis does not escape from the law laid down by Louis which generally governs the development of pulmonary tubercle, namely, that the apices of the lung are the first and the most profoundly altered by the neoplasm which extends from the apex to the base. The bronchi are always red and affected with catarrhal inflammation. The trachea, the lungs, and the first divisions of the bronchi generally contain granulations and even ulcers.

In the second case, namely, that of **acute pneumonic phthisis**, generalised granular phthisis is complicated in places by bronchopneumonia, more or less extensive patches of catarrhal or fibrinous pneumonia, acute pulmonary congestion, splenitisation, and atalectasia. Sometimes the whole of the lung, or the larger part of both lungs, is uniformly red, of the colour of red currant jelly, and congested to a degree reaching to splenitisation or diffuse apoplexy. On this red basis the small, opaque, grey or

white granules are plainly seen. Nuclei of catarrhal or fibrinous broncho-pneumonia are sometimes irregularly disseminated in the superior lobes; one or more small cavities are present in the apex, and intense congestion in the inferior lobes. In other cases extensive and recent catarrhal or fibrinous pneumonia, lobules of grey hepatisation, and diffused masses, composed of engorged or hepatised pulmonary tissue, may be observed along with numerous granulations; sometimes even caseous lobules may be found. This is the variety of generalised granular phthisis complicated with pneumonia which is called acute pneumonic phthisis.

These two varieties of acute phthisis differ as widely by their symptoms as by their pathology. In the first the general symptoms are the most prominent, while in the second the signs of capillary bronchitis, pneumonia, or pleural pneumonia are predominant. Sometimes at the post-mortem examination of patients who have died during convalescence from typhoid fever, scarlet fever, or from some accident, an eruption of miliary tubercle has been found, which had not been at all suspected.

b. **Subacute phthisis.**—In subjects who die after having shown, for a period of from three to six months or longer, the signs of galloping consumption such multiple and various lesions are found that two anatomical varieties of subacute phthisis may be distinguished. In one ulcerative broncho-pneumonia exists together with discrete or confluent tubercles of different ages, and cavities are rapidly formed; in the other slate-coloured interstitial pneumonia is found coinciding with fibroid granulations.

On examining the first variety by breaking down the lung throughout its whole extent, cavities are found in the superior lobe, about the size of a nut, communicating with the bronchi, and lined with a fusiform or pulpy grey layer, beneath which there is a highly vascular red zone. These losses of substance are surrounded by a zone of grey or yellow hepatisation, and the neighbouring pulmonary tissue is very red and slightly resistant. The surface of the bronchi is red, and covered with pus or muco-pus. The cavities correspond to the divisions of the bronchi, so that on section these tubes are seen slightly dilated and continuous with the anfractuous cavities which occupy the place of the terminal bronchi. The superior lobe is, moreover, scattered over with masses formed of confluent tubercles, which are surrounded by caseous pneumonia, and often ulcerated in the centre. The different lobes adhere together from pleurisy. The middle lobe of the right lung often shows cavities filled with pus, smaller and less

numerous than those of the superior lobe, and nuclei of confluent tubercles and caseous pneumonia disseminated in the midst of a congested tissue. In the superior part of the inferior lobe a few patches of more recent broncho-pneumonia and confluent tubercles are found, while at its base only discrete tubercles are found in the midst of pinkish or normally coloured pulmonary tissue. The visceral pleura, in places, or throughout its whole extent, is almost always covered with fibrinous false membranes, and shows a few semitransparent granulations. It is the same with the costal pleura. The pleural cavity contains a variable quantity of serous or seropurulent fluid. Though the two lungs are altered in a similar manner, one of them is generally more diseased than the other. What predominates and gives an anatomical character to this variety of phthisis is the suppurative, ulcerative, and rapid broncho-pneumonia which simultaneously disaggregates and softens a series of caseous nodules formed of tubercle and pneumonia.

In the second variety it is interstitial pneumonia which accompanies the tubercles. This disease is particularly met with in artisans who have worked in the midst of carbonaceous, siliceous, or other kinds of dust. On breaking up a lung one or more cavities, varying in size, are found in the apex, surrounded with slate-coloured interstitial pneumonia; nodules of caseous pneumonia are also sometimes found. At the base of the superior lobe hard nodules are often observed; they are about the size of a small nut, crepitate when cut by the scalpel, and show at their centre a group of granulations which are close together, semitransparent or yellow, pigmented at their circumference, and located in the midst of a hard, fibrous, and equally pigmented tissue. At the periphery of these patches interstitial pneumonia only is generally present; sometimes, however, discrete, smaller, and more recent tubercles are found. The bronchus which passes through one of these nodules is often inflamed and full of pus; a small cavity is sometimes being formed at the centre. The middle lobe, solidly attached to the superior and inferior lobes by fibrous thickening of the pleura, sometimes contains one or more small cavities and slate-coloured patches of interstitial pneumonia, sprinkled over with granules similar to the preceding, but generally smaller. In the superior part of the inferior lobe pigmented lobules of interstitial pneumonia are found also developed around a group of tubercles. Finally, at the base of this inferior lobe discrete tubercles are alone found, located in the midst of

congested tissue. Tubercular granulations, situated in a patch of interstitial pneumonia, are most frequently fibroid. The two lungs are affected in the same manner, or one of them is more diseased than the other. The visceral pleura is generally thickened and fibrous at the level of the hepatised lobe, and is closely adherent to the parietal pleura and the costal walls. The bronchi are acutely inflamed. In these two varieties of subacute or galloping phthisis the first divisions of the bronchi, the trachea, and the larynx are often the seat of tubercles and ulcers developed in the mucous membrane. The other organs are more rarely affected with tubercle than in acute phthisis.

c. **Chronic phthisis.**—The typical descriptions which we have given of the progress of the lesions in acute granular phthisis and in subacute or galloping phthisis is certainly subject to many variations. These variations are still more marked in chronic phthisis; in fact, nothing is so variable in nature and distribution as the pulmonary lesions in this form of phthisis. Tuberculosis may remain absolutely latent for a certain number of years when the lesions are limited. They are then reduced either to a few indurated nuclei formed of fibroid or pigmented granulations surrounded by interstitial pneumonia, or to a cavity enveloped in a dense layer in which fibroid granulations are found. These changes, which may be observed accidentally at the autopsy of old persons or of adults who have died from some other cause than phthisis, cannot sometimes be brought under the law of Louis, for they may be met with solely in the middle lobe, or even in the inferior lobe of the lung.

The most typical form of chronic phthisis is that in which the superior lobe is found, on dividing the lung, to be completely transformed into a series of large cavities communicating with the bronchi, and often with one another. These cavities are separated from the thickened pleura by a layer of fibrous tissue, and around them interstitial pneumonia is present with or without pigmentation, the whole lobe being in fact transformed into a callous tissue riddled with large holes. In the middle lobe the cavities are smaller, and nodules of caseous pneumonia and caseous tubercles are also found here. In the inferior lobe either small cavities are found in process of formation or discrete or confluent tubercles. The lung on the opposite side is less altered; the cavities are smaller, and hæmatosis can take place in a great part of its inferior lobe. Such lungs have been simultaneously or successively affected by a series of tubercular eruptions dating back from five

to ten years, and of which the evolution has been slow, but has never been arrested.

In other frequent cases of chronic phthisis the tubercles and cavities may exist for many years in one lung, or only in the apex of the two superior lobes, without any marked increase; then, at a given moment, an exacerbation occurs, which raises the disease in a few weeks or in two or three months to the condition of acute or of galloping phthisis; the lesions of both these forms of phthisis and old cavities will then be found.

In some cases of chronic phthisis slate-coloured interstitial pneumonia predominates, in others caseous pneumonia. Large cavities are generally observed in the parts affected with caseous pneumonia, but sometimes a whole lobe may be diseased without any cavities being found. The pleura is always altered in chronic phthisis. The upper lobes, surrounded by a fibrous sac, are adherent to the costal wall, so that to remove the lung from the thorax a thick, semitransparent and almost cartilaginous pleural tissue has to be divided. The pulmonary lobes are also almost always solidly united. On the external surface of the middle and lower lobes, at all points where there is adhesion or fusion of the two layers of the pleura, a more or less thick layer of fibrin is produced. The pleural cavity then contains a sero-fibrinous, sometimes purulent, fluid. The lesions of the pleura will be more fully considered later.

General views on pulmonary phthisis.—We place in the first rank tubercular granulations, and we subordinate to them all the other inflammatory lesions—congestion, engorgement, catarrhal, fibrinous, interstitial pneumonia, &c. We admit at the same time that these special inflammations belong by their course, their termination, and their pathology to the same general disease as tubercle. We consider tubercle to be a small disseminated neoplasm, which may be classified among tumours. It becomes caseous or fibroid as it grows old; it grows old and enlarges from the centre to the periphery. Tubercle often becomes generalised by affecting the neighbouring glands by means of the lymphatics and blood vessels, as do also other tumours. Secondary broncho-pneumonia is a most serious lesion, as it assists largely in the ulcerative process, the suppurative breaking down of the tissue, and the formation of cavities. Broussais has attempted to refer all the lesions of phthisis to simple inflammation; Cruveilher classed tubercles among inflammatory lesions, and later many pathologists, Lancereaux among others, regard them as the result

of a peculiar form of chronic inflammation. Laennec's opinion that tubercle was a parasitic and specific disease was long held by most physicians. The experiments of Villemin on the inoculation of tubercle, verified by a large number of observers, and acknowledged to be accurate by many of those who, like Cohnheim, contested them at first, have modified the views generally held on phthisis. They have reaffirmed the unity of phthisis, and tend to cause it to be looked upon as a chronic infectious disease, due to penetration into the lungs of infectious microbes, which are drawn in with the respired air (Cohnheim). Klebs and Reinstadler have described in tubercles an extremely minute organism, the *monas tuberculosum*, which are in the form of granules joined two or three together, and animated by very lively movements. Dogs and rabbits, inoculated with culture fluids containing this organism, have given some positive results. More recently Toussaint has cultivated a very small and immobile micrococcus, which is arranged in groups of three to ten, or in masses, and which he considers to be the parasite of tubercle. Still more recently Koch, by cultivating fragments of tubercle, has succeeded in isolating microbes which resemble the bacillus of lepra in shape, but are more delicate. He has succeeded in inoculating many animals with tubercle.

VI. The Pleura.

We have already described in detail the general pathology of the serous membranes (*vide* vol. i. p. 423); almost all hitherto said respecting the serous membranes in general applies to the pleura, and we shall hence only refer here to the lesions proper to it. In the majority of cases the lesions of the pleura are subordinated to those of the lung, and borrow from it their special characters.

Congestion, ecchymoses, hyperplastic pleurisy.—Congestion of the visceral pleura is always observed every time the lung is itself congested. The capillaries of the delicate layer of transparent connective tissue which forms the visceral pleura are filled and distended with blood; through this transparent visceral layer the interlobular septa of the lung can be seen, with the markings of the lymphatics and blood vessels. The polygons which the bases of the pulmonary lobules form on the surface of the pleura are bordered by a whitish, or in old subjects blackish, line, in

which the blood vessels, charged more or less with blood, and the lymphatics are easily recognised by the naked eye. The latter are at least as large as the interlobular veins, which are superficial and transparent, and having thin walls are flat. When the pulmonary congestion is very acute, as when dyspnœa is present, or cardiac or pulmonary disease or asphyxia due to any other cause (diseases of the trachea or larynx, drowning, strangling, &c.), small ecchymoses are found on the surface of the parietal pleura, which may measure many millimetres in diameter. These ecchymoses are characterised by extravasation of the red blood corpuscles into the connective tissue of the pleura, in consequence of distension of the capillaries. When recent they project slightly, and exude a blood-stained fluid on the free surface of the pleura. The extravasated blood corpuscles soon change into brown and black granules, and the red projecting patch flattens at the same time that it becomes grey-brown and finally black. In subjects who have died from cardiac disease accompanied with emphysema and acute dyspnœa numerous ecchymoses are generally found disseminated on the visceral pleura; some are red and recent, others dark brown, and others black or slate-coloured. Chronic congestion of the pleura is simultaneously observed; this consists particularly in more or less marked thickening of the membrane and in the formation of granulations or villosities composed of connective tissue. Under these conditions hydrothorax is rather frequent. The pleura is whitish and cloudy and has lost its transparency. This condition is, however, very slightly marked, and a certain skill is necessary to recognise it, for the altered visceral pleura is generally but slightly thickened and preserves its suppleness and brilliancy. The opacity of the pleura is due to thickening of the connective-tissue fibres, together with tumefaction and proliferation of the interposed cells. On carefully examining the surface of the pleura, particularly at the level of the anterior and sharp borders of the pulmonary lobes, small projections are often found resembling red fleshy granulations, large villosities, or filaments. These unite the edges and surfaces of the lobes which are in contact. These granulations and filaments are composed of connective tissue; they are supplied with blood vessels and are covered by epithelium (*vide* p. 80). These lesions, which are slowly produced, belong more to chronic pleurisy than to simple congestion; they attest to congestion accompanied with slight chronic inflammation.

Ecchymoses sometimes become the starting point of fibroid

growths; in fact, beside the small red or grey ecchymoses white prominences are found of the same form and size; they are fibromata composed of flattened lamellæ of connective tissue. These growths, which have been wrongly described as fibroid tubercles, and which Andral rightly distinguished from tubercular granulations, have indeed no structural resemblance to the latter. They exactly correspond to similar indurations of the fibrous capsule of the spleen and to the description given of lamellar fibroma (vol. i. p. 163); sometimes they are cartilaginiform in appearance. Chronic congestion and these slowly induced inflammatory changes may accompany fluid effusions into the pleura; this is generally limited to the lower part of the thorax. The fibrous false membranes, which unite the parietal

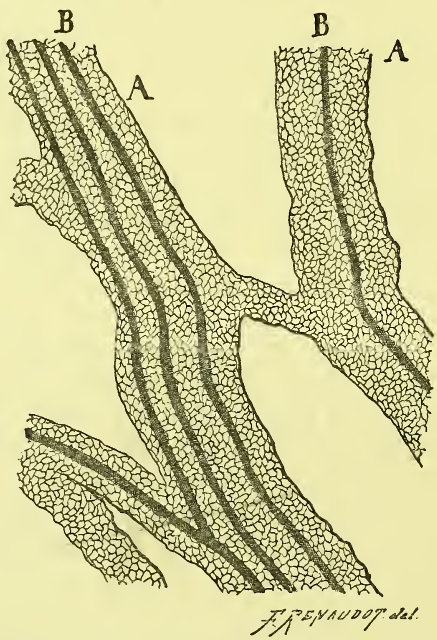


Fig. 80.—Filamentous Bands of the Pleura, covered by their Epithelium, which is plainly shown by Nitrate of Silver Staining.

b, blood vessels. Magnified 80 diameters.

pleura to the lung or the lobes to each other, are then œdematous; their vessels are distended with blood, and the connective-tissue fibres of the false membranes are separated by a fluid which contains large spherical cells and granular cells, as in œdema of the subcutaneous cellular tissue.

Fibrinous pleurisy.—In acute pleuritic inflammation fibrin is always formed on the free surface of the pleura; fibrinous flakes are often also found in the exuded fluid. These fibrinous exudations are found, for example, in purulent pleurisy, hæmorrhagic pleurisy, and in most forms of tubercular pleurisy; but what is called fibrinous pleurisy is more particularly that in which the inflamed membrane is covered by a layer of fibrin at the same time that the pleural sac contains a citron-coloured fluid which coagulates into a gelatinous mass. When this transparent fluid is withdrawn from the pleura by thoracentesis it is always found to contain a certain number of red blood corpuscles and lymph cells, and on examining the coagulated fluid under the microscope delicate networks of filaments of fibrin are seen. The fibrinous exudation is not produced at those points where the pleura is the seat of old fibrous growths, which hold the opposite surfaces of the serous membrane close together; still more is adhesion of the two pleural layers a positive obstacle to the deposit of fibrin. For it to be deposited the pleura should be, in fact, free throughout or for a great part of its extent, and should contain a more or less considerable quantity of fluid. As it is the upper lobes of the lung which have generally contracted adhesions with the costal pleura, pleurisy with effusion is generally formed in that part of the serous membrane which lines the inferior lobes. A sac thickened by false membranes is then formed by the costal, diaphragmatic, and visceral pleura, and filled with fluid. Fibrinous pleurisy is consecutive to pulmonary disease, or it is primary.

Very slight secondary fibrinous pleurisy always accompanies catarrhal, caseous, and acute lobar pneumonia, every time that the inflammation affects the surface of the lung. Thus in the dry, semitransparent, and grey hepatisation of fibrinous bronchopneumonia, which is so frequent in phthisis (semitransparent grey infiltration), a false membrane is seen on the surface of the pleura, which is sometimes extremely thin and transparent, and sometimes thicker and formed of several layers. On microscopic examination, the layers will be seen to be composed of a network of fibrin, the principal trabeculæ of which correspond to vessels situated beneath; the meshes contain tumefied endothelial cells and pus cells. Beneath this layer of fibrin the vessels of the pleura appear to be dilated, and the membrane itself is cloudy; it is either less transparent than normally or it is quite opaque and slightly thickened. This is due to the presence of

leucocytes between the connective-tissue fibres. The reticulated structure of a delicate false membrane is visible to the naked eye; if it is thicker its reticulated appearance is better seen and projecting trabeculæ are then observable (*vide* vol. i. p. 424). This fibrinous exudation is characterised by its friability, its sharp line of fracture, and its transparency. In pleurisy caused by the fibrinous pneumonia of tubercle the fluid exudation is generally scantier and citron-coloured.

In acute fibrinous or lobar pneumonia, fibrinous false membranes are always found adherent to the pleura if the hepatised part be superficial; but the quantity of serofibrinous fluid effused

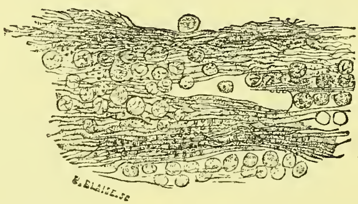


Fig. 81.—Section of Exudation of Pleurisy, in which Filaments of Fibrin and Pus Cells are found. Magnified 250 diameters.

into the serous cavity is small. The false membranes are in this case sometimes more opaque and yellower than in the preceding, when they coincide with the stage of suppuration or grey hepatisation in pneumonia; in their interior many granular lymph cells are found. On removing the fibrinous false membrane, dilated and prominent blood vessels are seen on the surface of the pleura in the form of granulation loops, and it is at the level of these newly-formed vessels that the exudation is thickest. The pleura is itself thickened and infiltrated with lymph cells. The parietal pleura may escape inflammation, but it is most frequently affected with the same lesions, though its circulation is quite independent of that of the visceral pleura. In order to explain the propagation of inflammation from the visceral to the parietal pleura, the quality of the fluid exuded by the former must be considered; everything leads one to think that this fluid irritates the surface of the parietal pleura at the same time that it causes a deposit of fibrin. Pleurisy related to pneumonia is sometimes accompanied with such an abundant effusion that the pleurisy becomes the predominant symptom; and later it may constitute the sole disease if it persists after the pneumonia is cured.

Idiopathic pleurisy, namely, that which occurs suddenly as

the result of chill, is far from being always alike; in fact, the character and quantity of the pleural effusion, the course and termination of the form of pleurisy, are very variable. It is always characterised by a layer of fibrin exuded on the pleural surface; it is almost always unilateral; generally the effusion is at its maximum on the eighth or tenth day; the quantity of fluid varies between one and three pints. Both the visceral and parietal pleuræ are congested at first, and they are immediately (that is to say, from the first day, as experiments on animals enable us to affirm) covered by a delicate layer of fibrin at the same time that fluid is effused into the pleural cavity. The effusion increases the succeeding days, and the deposit of fibrin thickens by the addition of fresh layers. Flakes of coagulated fibrin frequently float in the fluid, as may be seen when the fluid is withdrawn by thoracentesis. The visceral and parietal pleuræ are always thickened and less transparent than normally. In sections a large number of lymph cells are seen interposed between the connective-tissue fibres, which cells are more numerous as the surface is approached. The blood vessels are prominent and budding. The lymph vessels contain the same elements as the connective-tissue spaces, that is to say, lymph cells in great numbers, and often coagulated fibrin. The lung does not escape inflammation, and the superficial pulmonary alveoli are affected. Lymph cells are extravasated into these alveoli at the same time that their epithelium swells and becomes desquamated. It is hence seen that if pneumonia causes secondary pleurisy, primary pleurisy is always accompanied with superficial pneumonia, which passes unnoticed at an incomplete examination, but of which count must be taken, as Brouardel has shown, when considering prognosis and treatment. If, in fact, a fluid effusion is allowed to remain in the pleura, thickening of this membrane will be added to superficial inflammation of the lung, and will subsequently prevent the lung from expanding when the effusion is gradually absorbed.

Once fully developed, simple pleurisy undergoes **resolution** by itself, but generally very slowly if the physician does not interfere. This is what occurs during resolution: The effused fluid is gradually taken up by the lymphatics as soon as these become permeable again; the fibrinous false membranes undergo granular degeneration, as well as the leucocytes which have been extravasated either into the fluid or into the false membranes. The resorption of the exudation is impossible as long as the lymphatics

are obstructed with fibrin. An effusion very rapidly thrown out may disappear as rapidly and spontaneously. This is what occurs in pleurisy developed in the course of acute articular rheumatism; sometimes the fibrinous false membranes are themselves rapidly reabsorbed without leaving fibrous adhesions behind; but this is an exception. Generally the visceral and parietal pleuræ remain thickened. Under the layer of fibrin embryonic connective tissue is produced, and loops of newly-formed blood vessels project into the fibrinous false membranes which are interposed between the two layers of the pleura. These vascular trabeculæ are pushed out into the substance of the fibrinous false membranes, and the vessels join those coming from the opposite pleural surface; the filaments of embryonic tissue become organised, and an adult dense connective tissue is developed as the fibrin is broken down and reabsorbed. Thus when fibrinous pleurisy undergoes resorption slowly it terminates in producing organised and permanent filamentous or lamellar adhesions, varying in length, or a close union of the two layers of the pleura which remain adherent. The duration of this process is very variable. The slow absorption of the fluid and the organisation of the false membranes may be prolonged for a year or more. It is then said that the pleurisy, acute at first, has passed into the chronic condition.

In certain cases of simple pleurisy with serous effusion which may have lasted one or two months, and in which death has been caused accidentally by some other disease, the thickened visceral pleura is found at the autopsy to be covered with a thin layer of fibrin without the two serous surfaces being united by fibrinous or cellular false membranes. If, in a similar case, the effusion is abundant, the lung collapses and the visceral pleura, contracted and hard, keeps the lung in a permanently contracted condition. Sometimes a part of the lung, bound down by false membranes, projects in an irregular manner. On carefully incising the thickened pleura, so as to free the pulmonary parenchyma, it will be seen that it becomes inflated again, and takes its normal form. If a delicate section of a lung compressed in this way be removed and placed in water, the alveoli resume their normal form, and the alveoli will be seen, as we have already said, to be simply flattened together, without either their septa or their epithelium being sensibly altered.

Fibrinous idiopathic pleurisy may be accompanied with a very abundant and rapidly formed effusion. Generally the more abund-

ant the effusion the fewer fibrinous flakes there are in the fluid. This form of pleurisy with considerable effusion is sometimes produced at the commencement of tuberculosis in which pleural granulations are the first manifestation. The pressure exercised by such a considerable quantity of fluid causes displacement of neighbouring organs, particularly of the heart when the pleurisy is left-sided.

Hæmorrhagic pleurisy.—While the new membranes of the inflamed pleura are becoming organised accidents may be caused, due to the development of new blood vessels; the walls of these vessels being embryonic and very friable, the blood tension is sufficient to cause extravasation of red blood corpuscles, ecchymoses of the fibrinous false membranes, and finally effusion of blood into the pleural cavity. These accidents are sometimes found in idiopathic pleurisy, but they are then less marked; they are also occasionally seen in the more acute form of pleurisy which complicates subacute articular rheumatism. Their most frequent cause is pleural tuberculosis and pleuro-pulmonary cancer, and it is in these two diseases that hæmorrhagic pleurisy is most serious. The pleural cavity is then filled with blood contained between the superimposed layers of the false membranes, which are themselves red and composed of fibrin and vascularised embryonic connective tissue. Both the fibrin and the connective tissue are infiltrated by blood elements. If hæmorrhagic pleurisy is related to tuberculosis the embryonic new membrane contains tubercular granulations besides its numerous blood vessels. The extravasated blood undergoes the successive changes which end in the formation of hæmatoidin (*vide* vol. i. p. 45).

Another accident which sometimes occurs even in fibrinous pleurisy with serous effusion when acute is **suppuration.** It has been shown that between the filaments and laminæ, in the layer of fibrin which covers the inflamed pleura, a large number of lymph cells is always present; they infiltrate the superficial parts of the thickened pleura, and a large number are also always found in the transparent or slightly cloudy serous fluid which fills the pleural cavity. From causes not sufficiently understood simple pleurisy often becomes purulent after repeated punctures and when inflammation of the skin has been caused by the trocar. The introduction of pyogenic and infectious microbes probably plays an important part here. In a short time the lymph cells become extremely numerous in the fluid and false membranes; the fibrin is

disassociated; the effusion becomes cloudy and thick and takes the characters of serous or phlegmonous pus. This accident, which is rare in simple pleurisy, is much more frequent in secondary pleurisy. It is sometimes observed in pleurisy related to tuberculosis, albuminuria, gout, and almost always in that which follows infectious diseases, &c.

Purulent pleurisy.—Purulent pleurisy occurs spontaneously whenever metastatic abscesses, purulent foci, or lymphangitis are present on the surface of the lung, that is to say, in the infectious fever which may follow extensive injuries, large operations, or parturition; a local pulmonary lesion may also be the point of departure, as, for example, when lobular gangrene of the lung produces foci under the pleura, or when one or more small tubercular cavities are located superficially. These lesions are sometimes accompanied by purulent pleurisy even before the gangrenous or tubercular cavities open into the pleural sac; but the pleura suppurates still more when a cavity opens into it. Purulent pleurisy cannot be produced under these conditions if the pleura is thickened and indurated, for the new connective tissue hinders the progress of inflammation. Suppurative pneumonia may also cause a puriform effusion, but it is then slight. In the preceding examples purulent pleurisy is of pulmonary origin, but it is not always thus: abscess of the liver opening into the pleura through the diaphragm may also cause purulent pleurisy; and fracture of the ribs, complicated with perforation of the thorax, and abscesses related to caries of the ribs may also equally cause it. In some cases purulent inflammation of the lymph vessels on the surface of the lung has been noticed, and that form of dissecting pneumonia already described. The pus produced in septic pleurisy has a markedly fœtid odour even when there is no pulmonary perforation or gangrene. Cruveilhier relates having frequently observed gangrenous mortification of the parietal pleura in puerperal fever, or under other circumstances.

In empyema, paracentesis gives issue to a purulent fluid mixed with fibrinous flakes which are infiltrated with pus. After opening the pleural cavity, and washing it out with irritating or antiseptic injections, the serous membrane is in the condition of a large suppurating wound exposed to the open air: as would occur in such a wound, its visceral as well as its parietal layer becomes covered with fleshy granulations and is changed into a pyogenic membrane. The granulations become organised, approach one

another, and cicatrisation occurs by the union of the two opposite surfaces (as described, vol. i. p. 111). Purulent pleurisy is a disease of long duration, whether it be ultimately cured or whether it terminate by death. It sometimes ends by the spontaneous issue of the pus externally through an intercostal space. This termination is often preceded by ostitis, caries, or necrosis of one or more ribs; it is then announced by œdema of the skin. At other times the pus finds its way into the bronchi, or through the diaphragm into the peritoneal sac, or into the posterior mediastinum (Gendrin), or along the vertebral column as far as the psoas muscle. When a cavity or pulmonary abscess opens into the pleura, air penetrates with the pus, and pyopneumothorax is produced. Contraction and collapse of the lung then attain their maximum degree.

Chronic pleurisy.—Chronic pleurisy may occur spontaneously or follow acute pleurisy. Hyperplastic pleurisy (described on p. 168) is in reality spontaneous chronic pleurisy, characterised by the formation of connective tissue, which is first embryonic, then adult, and which terminates by producing fibrous thickening of the pleura, filamentous or membranous adhesions, or by causing complete obliteration of the pleural cavity. Spontaneous chronic pleurisy is also consecutive to slowly developed tubercular or cancerous pulmonary lesions. Chronic pleurisy following acute fibrinous pleurisy is characterised by the formation of fibrous tissue and organised false membranes. Such is its natural mode of cure. The definite organisation of false membranes often causes no accident whatever; this is the case when the fibrous trabeculæ or the thickened visceral pleuræ do not markedly hinder the expansion of the lungs or the movements of the ribs. But if after a considerable effusion absorption has been long delayed, the organised fibrous false membranes and the greatly thickened pleura hinder the normal play of the lungs in both inspiration and expiration. These false membranes solidly unite the visceral to the parietal pleura. Thus, as the fluid is absorbed, and the space separating the two pleural surfaces diminishes, the shrunken false membranes draw the costal wall towards the root of the lung. The thorax, which had before been dilated by the effusion, contracts; the ribs are drawn together and touch, particularly at the lower part, so that the diseased side becomes narrower than the healthy side; the shoulder drops, and the vertebral column shows a certain degree of

scoliosis with concavity turned towards the diseased side. After acute pleurisy, in which the pleura has become divided into chambers limited by thick false membranes, a more or less considerable portion of the liquid effusion may remain encysted in a fibrinous or fibrous sac. When the lesion is very old this pleural sac is often filled with a semifluid mass, composed of white blood corpuscles in a state of caseous degeneration ; its internal surface is lined with a very thick layer of fibrin, which is shining and transparent, and might at first sight be mistaken for an hydatid membrane, but microscopic examination settles the question. These fibrinous false membranes are adherent to the parietal pleura, which is thick, hard, fibrous, and sometimes calcified. In chronic interstitial pneumonia the visceral pleura is always much thickened. If it is a case of interstitial pneumonia without tubercle, or of induration of the lung around old cavities, or of dilated bronchi, or of any other process tending to induration of the lung, the pleural lesion is the same. The pleura forms a fibrous, dense, elastic, white or grey shell, slightly vascular, and from one-half to five millimetres, or even more, in thickness. This inextensible tissue is formed of thick and close fibrous fasciculi, with flat connective-tissue cells, and contains blood vessels with dense and rigid walls. In some parts it is often œdematous, but this œdema does not differ from that of ordinary connective tissue. Fibrous induration of the pleura is generally, like the primary lesion which caused it, situated at the apex of the lung; it extends from the lung to the ribs, and causes complete union of the two serous surfaces. It is often impossible to remove the lung except by peeling off the periosteum from the ribs, and by cutting away the diseased pleura at their level. When one has succeeded in detaching the pleura and lung, white bands may be seen marking the situation of the ribs on the external surface of the parietal pleura, the thickened periosteum being continuous with the newly-formed connective tissue. The bony part of the ribs is sometimes affected at the same spots with condensing ostitis. More or less extensive calcareous plates are sometimes found in chronic pleurisy. They form a cuirass either on the surface of the lung or on the parietal pleura. When suppurative pleurisy has passed into the chronic condition, it is acccompanied with lesions similar to those just described. There may be also caseous degeneration of the pus, chambers formed by pseudo-membranes, fibrous thickening of the pleura, and lesions of the ribs, such as exostosis, periostitis, necrosis, &c.

Tumours of the pleura.—The most frequent tumours of the pleura are tubercular granulations. When they are few and recent they only cause slightly marked pleuritic inflammation, but when more numerous and older they always cause one of the forms of pleurisy, so that in studying pleural tuberculosis one cannot do otherwise than pass in review the various forms of pleurisy. The granulations commence either on the surface of the visceral pleura or in the false membranes which unite the visceral to the parietal pleura, or in the costal pleura itself. Thus, in certain cases, in which the pulmonary granulations are not so marked nor recent as to be characteristic to the naked eye, perfectly recognisable tubercles are often found on the parietal pleura. As adhesions between the apex of the lung and the costal wall are then constantly present, we may conclude that the tubercles are propagated by continuity of the tissues or by means of the lymph vessels. The granulations cause vascularisation and ecchymoses at their periphery, an exudation of fibrin on their surface, a new formation of embryonic tissue around themselves, and finally an effusion of fluid into the serous sac. Numerous and recent granulations sometimes cause a pleurisy characterised by a delicate false membrane on the surface of the pleura, and by very abundant serous effusion. This almost constantly occurs in general tuberculosis especially affecting the serous membranes, a form which has been well described by Empis under the name of *granulie.* Older tubercular pleurisy is characterised by numerous granulations on the surface of the serous membrane, in the pseudo-membranes formed of embryonic tissue and fibrin, and in the serous membrane itself, which is changed into a semitransparent embryonic tissue. This altered parietal pleura may be so thick as to be many millimetres, or even a centimetre, in diameter. In a section cut perpendicularly to the surface, the tubercular granulations are seen to form many layers ; their opaque centres are very distinct. As we have already said, hæmorrhages may occur from the false membranes and an effusion of blood into the serous cavity. In tuberculosis, purulent pleurisy is almost always related to the presence of recent superficial cavities, or to perforation of the lung.

Fibroma of the pleura is represented by small growths and the fibroid granulations already described (p. 170). A rather large quantity of adipose tissue may be present in some of the polypiform growths without their meriting the name of lipomata.

Carcinoma of the pleura is very often developed after primary

or secondary carcinoma of the lung, or in consequence of carcinoma of the breast. In the latter case the lesion is propagated by direct infection, and carcinomatous nodules are thus produced in the pectoral and intercostal muscles and in the parietal pleura. On the surface of the visceral pleura the lymph vessels are then observed to be inflamed and changed by the carcinomatous neoplasm. These vessels may be affected partially or generally; their lumen is occupied by a white or yellowish mass, more or less solid, composed of polygonal or spherical epitheloid cells, and their walls may sometimes be seen to be infiltrated with carcinomatous granulations. In scirrhus the cancerous granulations of the pleura are small, hard, or united into isolated patches; when encephaloid in character they are generally larger, more prominent, and centrally depressed. These nodules, when located in the parietal pleura and in the deep layers near, press upon and interfere with the intercostal nerves, causing intercostal neuralgia. The different varieties of carcinoma, scirrhous, encephaloid, colloid, melanic, &c., have been observed in the pleura.

Pavement epithelioma has been seen in the pleura as well as in the lung consecutive to epithelioma of the skin. **Sarcoma** and **chondroma** have also been observed there.

Section II.—The Digestive System.

CHAPTER I.

THE MOUTH.

I. Normal Histology of the Buccal Mucous Membrane.

The buccal mucous membrane, which is directly continuous with the skin, is composed, like the latter, 1st, of an epithelial investment; 2nd, the corium, which is a tissue composed of connective-tissue fasciculi and elastic fibres, and provided with papillæ and glands; 3rd, of a layer of loose connective tissue by means of which it is united to subjacent parts. The mucous membrane, properly so called, or the mucous chorion, is directly continuous with the derm of the skin. On its external surface there are numerous papillæ similar to those of the skin; they are simple or composite, and are provided with nerves and vessels. On the tongue they show special characters, but in all other parts of the mouth they are not seen at first sight, for they are hidden under the epithelial lining. The epithelium is of the stratified pavement variety; the lower cells are cylindrical and implanted perpendicularly on the surface which they cover, on the papillæ as well as in the interpapillary spaces; above these is a layer of polygonal cells with blunt angles, and in the most superficial layer the cells are flattened parallel to the mucous surface. All the cells are united together by filaments, which are as well marked as those of the Malpighian layer. The cells are produced unceasingly in the deep layers from the cylindrical cells, the division of which is preceded by that of their nucleus; they are constantly changing, just as the cells of the epidermis. The mucous chorion adheres closely to the bony framework of the gums at the level of the neck of the teeth, and also to the palate; on the back

and edges of the tongue muscular fibres are inserted into the mucous membrane and prevent gliding movements. In other parts of the mouth, the lips, the frenum of the tongue, the cheeks, it is slightly mobile, and, owing to its lining of cellulo-adipose tissue, may be slightly displaced. The glands of the mucous membrane are acinous, round in shape, and measure from 1 to 5 mm. in diameter. They are found on the posterior surface of the lips, on the mucous membrane of the cheeks, on the hard and soft palate, at the base of the tongue behind the lingual V, beneath the follicles at the base of the tongue, and at the level of the circumvallate papillæ. Near the tip of the tongue, on its inferior surface, they form two elongated glandular patches, the excretory ducts of which open on each side of the frenum; these are called the glands of Blandin and Nuhn. The excretory ducts of these various glands are formed of connective tissue lined with cylindrical cells. They terminate in glandular vesicles, pyriform or spherical in shape, and are generally lined with mucous cells.

In man, three kinds of papillæ are distinguished: the **circumvallate papillæ** which form the V at the back of the tongue; they are composed of a large central papilla with a deep base; this is surrounded by a kind of ring or rampart, which is encircled again by a groove, the two lips of which are lined with epithelium, not entirely pavement in character. Into this groove special glands open. On the external surface of the central papilla and on the corresponding surface of the rampart groups of sensorial epithelial cells are found, which have been called the organs of taste. These organs were discovered by Loven and Schwalbe, almost simultaneously. They are cylindrical and elongated cells with an oval nucleus near the centre. They may be divided into a peripheral portion which terminates in a rod at the surface and a deep or central portion beyond the nucleus; the latter is continuous with a nerve fibril. The **fungiform papillæ** are distributed over the back, edges, and tip of the tongue. They contain organs of taste, but they are not as numerous nor as regularly arranged as in the circumvallate papillæ. The **filiform papillæ** do not contain them; they are covered with stratified epithelium similar to that which lines most of the buccal cavity. At the edges of these papillæ the superficial lamellar layer of the epithelium forms long simple or fringed filaments; the most superficial epithelial cells, those about to undergo desquamation, are the seat of an abundant growth of a fungus called the *lepto-*

thrix buccalis, which is found in the mouth of almost every person. The different papillæ of the tongue contain vessels and nerves; the latter are very abundant in the papillæ which contain organs of taste, in the interior of which they seem to terminate almost entirely. The buccal mucous membrane also contains numerous lymph follicles, which are the simple follicles found at the base of the tongue from the border of the lingual V to the epiglottis; the tonsils are also nothing else than composite follicles. The simple follicles are composed of a round projecting body, at the level of which the mucous membrane, though depressed, has its various epithelial layers. The follicles, which are formed of an adenoid tissue, are enclosed in a lymphatic sinus at which afferent lymph vessels arrive and from which efferent vessels depart. The lymph vessels of the mucous membrane are very numerous.

II. Pathological Changes of the Buccal Mucous Membrane.

Stomatitis.—Stomatitis is seen under forms which vary greatly according to the degree of inflammation, the cause, the depth of the layers affected, and the part which is diseased. We shall describe these varieties successively.

Superficial stomatitis is often marked, besides the redness of the mucous membrane, by small white patches which are seen on the posterior surface of the lips, the alveolar mucous membrane, and the soft palate. These patches, frequently seen in smokers of cigars, are sometimes observed in typhoid and other fevers, and are constant at the commencement of mercurial stomatitis. They are due to the presence in the superficial layer of the epithelium of a fluid exudation containing lymph cells which have been extravasated from the vessels of the papillæ, and also to tumefaction of the epithelial cells. The epithelium has thus lost its homogeneity, and consequently its transparency. These patches must not be confounded with those of diphtheria, which are much thicker, more opaque, grey and dull, and which, instead of blending with the epithelial lining, rest on the surface of a more or less swollen mucous membrane.

The white patches of **mercurial stomatitis** are very marked and rather extensive. They are opaque, which is due to the fact that large numbers of pus cells are infiltrated between the epithelial cells. This variety of stomatitis frequently causes superficial ulcerations. It commences by redness and swelling of the mucous

membrane of the gums, and extends to that of the cheeks, lips, and tongue; on the latter the teeth make indentations and may even cause superficial erosions. A more or less abundant salivation is then established. In very acute mercurial stomatitis, induced during the treatment of syphilis, frequent formerly, but rare nowadays, patients have excreted many quarts of saliva in the twenty-four hours; the swollen and inflamed tongue sometimes projects in front of the teeth. This is a condition of more or less acute inflammation of the mucous membrane implicating both the epithelial and the submucous layers.

In **the stomatitis of typhoid fever**, round ulcerations, from 1 to 3 mm. in diameter, are sometimes observed on the internal surface of the lips and tongue. On microscopically examining sections of these ulcerations, it will be seen that at these spots the epithelium of the mucous membrane is shed, and the superficial layer of the mucous tissue destroyed. The base and edges of these ulcerations are composed of mucous tissue infiltrated with lymph cells, which change may be traced to some extent around the ulcerated point. In typhoid fever the vessels of the villosities and papillæ of the tongue will be found in delicate sections to be greatly distended with blood; sometimes lymph cells are found extravasated around them. In acute diseases, particularly in typhoid fever, the tongue is dry, and though red at its tip and edges it is black and cracked at the middle. Its dryness is partly due to the fact that the patients lie unconscious or asleep with their mouths open. The inflamed mucous membrane is covered by numerous layers of epithelium, which are accumulated on the surface of the filiform papillæ. The thick crust on the tongue is composed of these cells. The black colour is due to the presence of small foreign bodies which have entered the mouth with the air. Sometimes, however, this colour is produced by the extravasation of red blood corpuscles from the vessels. Between the papillæ cracks are often observed containing mucus, leucocytes, and red blood corpuscles. In typhoid fever the soft palate is equally congested, of a deep red colour, smooth, dry, or covered with adhesive, stringy mucus. The morbid buccal mucus is thick, and more or less opaque and stringy, and that which the patients eject is characteristic of this form of stomatitis.

Stomatitis due to special causes has characteristics peculiar to itself. Thus in **labial or buccal herpes**, which shows itself in the form of vesicles on the skin of the lips, it may also commence by vesicles on the internal surface of the lips and cheeks and on the

soft palate. But the vesicles do not last so long in the mucous membrane as they do on the skin; there, in fact, the epidermal layers raised by the effused fluid resist strongly, while the more delicate opaque epithelial lining is more easily broken down, and falls, leaving in the place of the vesicle a small ulceration, which persists for some days.[1]

The **lead line**, which is seen sometimes on the buccal mucous membrane around the neck of the teeth, is violet or black, and is produced by the presence of fine metallic granules in the cells of the deepest layers of the epithelial lining. These metallic particles, acting like foreign bodies, often cause more or less marked inflammation of the gums.

Eruptive fevers reveal their presence in the buccal mucous membrane by an eruption similar to that of the skin in measles, by red papules and macules easily seen on the soft palate; in variola by pustules seated on the tongue, soft palate, internal surface of the lips and cheeks; in scarlatina by a diffused redness principally seen on the tongue, soft palate, and tonsils. In serious cases of scarlatinal stomatitis the redness is often followed by desquamation of the epithelium in the form of soft, white, pultaceous membranes—pultaceous angina. When the epithelium of the tongue is desquamated, the lingual mucous membrane becomes red, shining, and smooth, looking as if it had been varnished. This desquamation is quite characteristic of scarlatina.

Diseases of the skin, such as eczema, pemphigus, and erysipelas, are sometimes accompanied, the latter particularly, by a similar eruption of the mouth and pharynx. This eruption is vesicular in eczema, is in the form of bullæ in pemphigus, and is shown by intense redness with mucous secretion, or with dryness of the mucous membrane, and sometimes with elevation of the epithelium in erysipelas.

Among cases of chronic stomatitis related to cutaneous disease, we find a local lesion of the mucous membrane, which is described by Bazin under the name of **buccal psoriasis**, and **ichthyosis** by the English. This lesion is characterised by white mammillated patches, situated on the tongue or lips, the mucous membrane of which is often split or cracked. In two cases examined by Debove ('Thèse de Doctorat,' 1873) the epithelial layer was slightly thickened, and the nuclei of the cells had become vesicular at the level of the psoriasic patches. The hypertrophied and condensed

[1] For the structure of vesicles and pustules see the description given of them in the chapter on the Skin.

mucous tissue had attained a thickness four or five times greater than normally; there was here an actual sclerosis of the mucous membrane; the connective tissue was infiltrated with lymph cells, and the lingual papillæ had taken a form resembling that of the cutaneous papillæ. The fibrous tissue dipped deeply between the muscular fibres of the tongue, causing atrophy of the superficial muscular fasciculi. The cracks observed in this disease correspond to the normal grooves and folds. These lesions are rather frequently seen near epithelioma of the tongue.

A subacute or chronic localised inflammation characterised by swelling of the mucous membrane of the gums is produced rather often at the spot where carious teeth pierce the mucous membrane. **Scorbutic stomatitis** is recognised by acute congestion of the mucous membrane and a tendency to hæmorrhage. Around the teeth the mucous membrane of the gums becomes fungoid, and shows vascularised fleshy granulations, which bleed easily.

The syphilitic lesions of the buccal mucous membrane are **mucous plaques**, and the deeper ulcerations which follow gummata. Recent mucous plaques are recognised by the white, opaque, or opalescent colour of the superficial layer of the epithelium, an appearance due to the swollen and cloudy condition of the epithelial cells and to migration of the lymph cells. If the mucous plaques are old they cause thickening of the mucous membrane and submucous tissue, and they then project slightly; if they do not project in a marked manner they can nevertheless be appreciated by the touch by a certain hardness and thickness of the mucous membrane. They are generally seated at the edge, tip, and back of the tongue, on the tonsils, at the corners of the lips, where the mucous part of the plaque is white, while the cutaneous portion is covered by a crust. They may also appear in all other parts of the mouth. The structure of mucous plaques of the mouth will be described in the chapter on lesions of the tonsils. More deeply-situated syphilitic lesions begin by tumefaction of the submucous tissue, and by deep tubercles or gummata which soon undergo ulceration. They are found on both the hard and soft palate, the tonsils, and the tongue. Gummata of the soft palate commence by tumefaction with induration of the connective tissue and consequent rigidity (Fournier). They often cause perforation by means of which the mouth communicates with the nasal fossæ.

Tertiary glossitis is divided by Fournier into sclerous glossitis, which may be superficial or deep, and gummatous glossitis, which

is sometimes superficial, sometimes deep, that is to say, intra-muscular. Superficial sclerous glossitis is characterised by limited thickening and induration of the mucosa, circular or oval in shape, bright or dark red in colour, located in the midst of a mucous membrane which is dry, smooth, and shiny. In sections the cellular part of the mucosa seems to be thick, fibrous, and infiltrated with small lymph cells; the papillary layer is not much altered, but it is smothered under a flat epithelium which the epithelial filaments of the filiform papillæ can no longer succeed in raising; whence mucous plaques seem to have no papillæ. Deep sclerous glossitis seated in the muscular layers of the tongue causes hypertrophy of the whole organ, and generally prominent nipple-like projections on its dorsal surface. The lobules are separated by grooves 1 to 3 mm. deep. In both these forms of sclerous glossitis there are no deep ulcerations, but simply accidental erosions due to the contact of irritating bodies. Superficial gummata of the tongue are developed in the mucosa or corium; they are small, about the size of a pea or cherry; sometimes they are solitary, sometimes several are grouped together. They commence by an induration, which soon softens and opens spontaneously. These have the inverted-funnel shape characteristic of all gummata, with the opening narrower than the base. Deep-seated gummata of muscle are much larger than the preceding, and are found in the lingual muscle, impinging on the connective tissue of the mucous membrane. They are often as large as a nut, and sometimes as many as four to seven are found. They open by an orifice which is narrow at first, but which gradually increases in width.

Ulcero-membranous stomatitis is characterised by diffuse infiltration of the corium by pus and fibrin. The circulation is arrested in the capillary vessels, which are compressed by the exudation, and the morbid part undergoes the eliminative ulceration which follows all mortification. Ulceration invades the deep and superficial layers of the mucous tissue; the edges of the ulcer are sloping, and its base is grey or black, blood-stained, fœtid, and covered by a grey opaque débris. On washing the base of the ulcer irregular filaments are detached, formed of elastic fibres; connective-tissue fibres and altered blood vessels are also dissected out by suppuration. The ulcers are generally seated on the internal surface of the lips and cheeks, on the gums, and sometimes on the soft palate and tonsils.

Diphtheritic inflammation is characterised by the presence of

white opaque false membranes, which vary in thickness, are rather dense, and are adherent to the surface of the mucous membrane (*vide* vol. i. p. 103). They are found chiefly on the tonsils, the soft palate, and its pillars. At its commencement small white spots are observed, which, by extending and blending together, form a dense pseudo-membranous patch, which is adherent to the mucous membrane which it covers. The first false membrane may be raised and detached artificially, but a new one is soon produced. Sometimes these false membranes are arranged in strata, the oldest of them being pushed up by the new as the latter are developed on the surface of the mucous membrane. The uvula may thus be covered and surrounded by these growths (for the histological details see 'Diphtheritic Tonsilitis,' described in the following chapter.) Serious angina, and particularly diphtheritic angina, is sometimes accompanied with paralysis of the soft palate, which may be followed by paralysis of other parts of the body. According to Charcot and Vulpian the nerve tubes of the motor nerves of the soft palate have then undergone morbid change, and show granular degeneration of their medullary sheaths.

Gangrene of the mouth or noma follows infectious diseases such as measles, small-pox, gangrene of the lung, &c. It first commences in the deep layers of the buccal mucous membrane, progressively invades all the subjacent layers, and often terminates by fistulæ of the cheeks or by destructive suppuration which may affect even the blood vessels of the part, and the external carotid may be opened by ulceration.

Tumours.—Persistent hypertrophy of the tongue and some of the parts which form the wall of the mouth is not so very rare. Hypertrophy of the lips, soft palate, and tongue is observed in scrofulous subjects and in cretins. Congenital hypertrophy of the lips—**macro-cheilia**—and of the tongue—**macro-glossia**—consists in thickening with new formation of connective tissue; the lymph vessels undergo at the same time a very remarkable dilatation, and form cavernous spaces. These spaces are lined with an endothelium, and are filled with serum containing numerous lymph cells (Virchow, Billroth). These lesions are similar to those found in elephantiasis. In scrofulous subjects also, hypertrophy of the soft palate may be observed which is caused by a new formation of connective tissue accompanied by considerable hypertrophy of the acinous glands of the part. The soft palate

may attain 1 c.m. in thickness. The hypertrophied glands preserve their usual form and normal structure.

Cysts.—Cysts which have their point of departure in the ducts or saccules of the glands are rather common in the mouth. Besides those already mentioned (*vide* vol. i. p. 297) they are frequently met with in the hard palate of new-born infants. Developed in the excretory ducts of the mucous glands, they are seen in the form of small, round, white or yellow grains, containing flat, lamellar epithelial cells, similar to those of the mouth (F. Guyon and E. Thierry, 'Arch. de Phys.' 1860).

Ranula.—Ranula is a cystic tumour formed in the floor of the mouth. It is generally round or oval, about the size of an almond or nut, unilateral, its anterior extremity extending as far as the frenum of the tongue; it sometimes impinges on the opposite side, when it appears to be bi-lobulated. If it is very large it pushes the tongue back, projects into the submaxillary region, and becomes indented by the teeth. The tumour is covered above by the buccal mucous membrane, which is thinned and but slightly adherent; it rests on the myo-hyoid and hyo-glossus muscles, to which it is closely adherent. It is caused by dilatation of an excretory duct of one of the sublingual glands. Such is the seat and configuration of classic ranula, which must not be confounded with cystic tumours of the floor of the mouth of quite another origin, but which are also called ranulæ by many surgeons. For a long time ranula was regarded as being always produced by the accumulation of saliva in Wharton's duct, but the surgeons of this century, Dupuytren, Malgaigne, Robert, Broca, Follin, and Duplay, &c., have given a description of it which has been recently perfected by Recklinghausen. It is particularly on the researches of the last-named that we base the following description of the histological characters of ranula, after which we will indicate the cystic tumours of the floor of the mouth for which it may be mistaken.

The fluid contained in the cyst seems to be viscous, stringy, and resembles a solution of sugar or gum, or rather uncooked white of egg; sometimes it is of a brown or reddish tint. It contains 95 per cent. of water, mucin, and sodium-albumin, but no sulphocyanide of potassium, nor any ferment capable of changing starch into sugar; but in it are found polymorphous epithe-

lial cells, colloid cells, and hyaline globes. If the internal surface of the cyst be stained with nitrate of silver, as soon after removal as possible, two layers of cells are seen: 1st, a superficial layer of large cylindrical ciliated cells, 13 μ to 18 μ in diameter, with oval nuclei; 2nd, a deep layer of small polygonal cells, measuring 5 μ to 7 μ in diameter, with a rather large round nucleus. The inferior extremity of the former passes between the second. In some parts of the cyst the cylindrical cells are smaller and are not ciliated. The cells do not become caliciform and contain no mucus, which indicates that the cyst wall takes no part in the secretion of the fluid. In a case of which Recklinghausen gives a detailed account, the orifice of the duct was found at a point in the internal wall of the cyst where the sublingual gland persisted, and he was able to pass in a bristle. The presence of glandular lobules in the wall of the cyst (Robin) is a fact greatly in favour of the view that ranula develops from the duct of a gland, but it is still more clearly demonstrated when the gland can be reached by a duct which opens itself into the cyst. In the wall of the cyst a hyaline layer resembling a basement membrane may often be found beneath the epithelial cells, and in contact with them. The rest of the wall, which varies in thickness, is composed of fibres and lamellæ of connective tissue, elastic fibres, and blood vessels. Besides this fibrous layer muscular fasciculi and nerves are found, which shows that the tumour has developed in the tongue itself and not in one of the salivary glands; for there are, in fact, no muscles in the floor of the mouth except the lingual muscles. Finally, the upper part of the tumour is covered by the visceral mucous membrane. The cyst is not always simple; there are sometimes many swellings or diverticula intercommunicating; when it passes under the frenum of the tongue it appears to be bi-lobed. Ranula is very rarely double, but it is possible that the ducts of the two sublingual glands may be simultaneously or successively dilated. Cases of congenital ranula, or which have appeared a few days after birth, have been described. Jobert, Stoltz, &c., have mentioned cysts of this kind seated in the sublingual gland; but there are, on the other hand, congenital cysts due to dilatation of Wharton's duct (Guyon, Lannelongue). Many observers have related cases of acute ranula, that is to say, of tumours which have very rapidly attained a considerable size. They have attributed this rapid development either to rupture of Wharton's duct or to its sudden dilatation, or to inflammation set up in the floor of the

mouth after ranula had already commenced. These cases still require elucidation.

The location of classical ranula in a duct of the sublingual gland is well established not only by the cases given, but also by the fact that Wharton's ducts are generally easily sounded with a stylet, when a few drops of saliva may be extracted by the ostium umbilicale on exciting the salivary secretion. The other ducts of the salivary glands are equally free. To understand how ranula develops we must, Recklinghausen says, consider how other cysts seated in glandular ducts are produced, more particularly those of the vulvo-vaginal glands. They are due to chronic inflammation, with sclerosis of the connective tissue surrounding the glandular ducts, whence results narrowing of these ducts to a point, and the secretion continuing, dilatation is produced behind the stricture. Fibrous myxangitis is thus set up, primarily affecting the glandular duct. It is, in fact, much easier to understand the dilatation of a duct surrounded with loose connective tissue than distension of the glandular saccules. The latter are, however, often filled with mucus. The duct of the gland being gradually dilated, its wall becomes thickened by the neighbouring tissues, fibrous, muscular tissue, &c. The epithelial lining of the internal wall of the cyst thus formed contains cylindrical cells which, it is true, differ from the normal cells of the duct by being ciliated.

The cystic tumours, to be distinguished from ranula, are those resulting from dilatation of Wharton's duct, or duct of the submaxillary gland; the serous cysts of cellular tissue or of the mucous bursa of Fleischmann, which are, moreover, very rare; and congenital, multilocular cysts similar to those in the neck. Finally, hydatid cysts may be found in the floor of the mouth (Gosselin and Laugier), and effusions of blood (Dolbeau). Dermoid cysts are very rarely found in this region.

The different varieties of **sarcoma** are very common in the gums and in the maxillary bones. The small tumours called **epules** are myeloid or ossifying sarcomata (vol. i. p. 142). Sometimes small, hard, round and sessile **fibromata** are developed in the connective tissue of the mucous membrane of the tonsils. Fibrous polypi, springing from the basilar process, may spread in the most diverse directions and enter the mouth by the nasal fossæ, for example, after having perforated the hard palate. On the internal surface of the lips and on the tongue **angiomata** may be found; they are seen during life in the form of many sub-

mucous tumours of different sizes, and sometimes as small superficial serous cysts projecting under the surface of the mucous membrane. These cysts, which must not be confounded with glandular cysts, contain a transparent fluid. The tumours examined microscopically in sections cut perpendicularly to the mucous membrane show cavernous tissue at their base, the lacunæ of which are filled with blood. The superficial serous cysts are nothing else than the lacunæ of the cavernous tissue situated in the papillæ and projecting under the surface. They are filled with fluid containing fine granules and a few red blood corpuscles, and are limited by a thin layer of fibrous tissue and by the thinned epithelium of the mucous membrane. Their internal surface is lined by a layer of vascular endothelium. Beside them are seen papillæ with dilated blood vessels intercommunicating, and which end in the formation of a cavernous tissue. The lacunæ of this tissue finally become isolated, cease to intercommunicate, receive blood from the general circulation, and become true serous cysts. Angiomata are often present at the free border, or at the commissure of the lips (*vide* vol. i. p. 242). **Lipomata** are found under the mucous membrane of the cheek, and sometimes at the tip of the tongue and on the posterior part of the lips.

Epithelioma, pavement, lobulated, horny, or mucous, is frequent on the lips; it is met with also in other parts of the mouth, in the tongue in particular.

Labial cancroid is almost always seated on the lower lip. It generally begins at the juncture of the mucous membrane and the skin in the form of a small warty unequal excrescence, which is soon covered by a blackish-grey crust. If the tumour at its commencement be examined microscopically in sections cut perpendicularly to its surface, it will be observed that the papillæ are hypertrophied, and that the interpapillary mucous body has sent out buds which dip down more or less deeply into the derm. These epithelial buds undergo a different epidermal evolution to the normal, in that the cells, instead of undergoing horny change when they reach the superficial layers, accumulate at certain points in the form of epidermal globes. These epidermal buds undergo, in consequence of the active growth of which they are the seat, partial disintegration, and the repeated products thrown off on the surface form the crusts which cover the warty growths of cancroid. At the moist parts of the lips the disintegrated cells do not dry, and form a white mass which is easily detached.

Sometimes the hypertrophied papillæ become enormous. Covered by the dried crust, they may form a prominent, hard, and irregular crust, in fact a kind of horn, which may be a centimetre in length. When the new growth is situated in a region which contains hairs and sebaceous glands, the epithelium of these glands and that of the external root-sheaths of the hairs concur in the development of the neoplasm (*vide* vol. i. p. 264). When the tumour extends and becomes deeper, ulceration always occurs; the floor of the ulcer is granulating, unequal, blood-stained, and partly covered by a crust similar to that just described, in which disintegrated epithelial cells are mixed with pus and blood. The edges of the ulceration project and form the firm, almost cartilaginiform border which characterises all ulcerating epitheliomata. Sections of this border show that it is produced by hypertrophy of the papillæ and budding of the interpapillary epithelial tissue, similar to that which characterises the commencement of the tumour. The neoplasm gradually invades the deeper parts, while the ulcerative process of which it is the seat tends to destroy it, without ever doing so completely, except in very rare cases. Finally, the muscles of the lips are partly destroyed and the inferior maxilla is itself invaded. The lymphatic glands of the submaxillary region contain lobules of epithelioma similar in structure to the primary tumour.

Epithelioma of the tongue is generally tesselated, lobulated, mucous, or colloid (*vide* vol. i. p. 257). Its development is much more rapid than that of the lips, and it is quite exceptional for its extirpation not to be followed by a rapid return. It ulcerates from the commencement. The base of the ulcer is papillary, villous, uneven, bleeding, pulpy, and its edges present the characteristic border. In sections passing through the ulceration and the subjacent parts it may be seen that lobules of epithelioma extend into the muscles and there produce the morbid changes described above (*vide* vol. i. p. 265).

Tubular epithelioma is frequently developed in the superior maxilla, the hard palate, and the maxillary sinus, where it forms masses composed of round, unequal, soft, friable, vascular buds. In sections the morbid tissue seems to be formed of a stroma generally rich in cells and which limits irregular anastomosing tubes; these are filled with epithelial cells which do not show epidermal evolution (*vide* vol. i. p. 269). These tumours develop very quickly, and always return if removed.

Tubercles of the buccal mucous membrane have been studied of

recent years by Ricord, Juliard, Trélat, Féréol, Thaon, Spillman, &c. They commence by one or more small granulations situated on the surface of the mucosa, and they soon become opaque and yellow centrally. They are usually formed on the edges and tip of the tongue, but they may also appear at the base of this organ, on the soft palate, and even on the lips. In the lingual mucous membrane the new growths commence by one or more confluent granulations situated on the surface of the mucosa, where they form a slight relief. Sometimes an erosion is observed, limited at first to a tubercle, the cuplike base of which contains a little caseous pus. If the process be followed day by day, the discrete and superficial tubercles may be observed to become entirely eliminated; a spontaneous cure is then the consequence. More often the ulceration occurs in a group of granulations. The ulcer becomes irregularly hollowed, and its edge festooned, for each primitive granulation is destroyed separately. The edge of the ulcer is thick and generally budding, and under the microscope nothing is seen but embryonic tissue and fleshy granulations. As the ulcer grows in depth and extent the neoplasm invades the deeper parts. The tubercular ulcer progresses slowly. Sometimes it retrogrades and seems to improve under local treatment.

On examining sections of an ulcerating tubercular tumour of the tongue under the microscope, with a low power, the edges of the ulcer are seen to be formed of the lingual mucous membrane, which is much thickened, granulating, and covered with epithelial layers. The base of the ulcer shows buds composed of embryonic tissue. In the connective tissue of the mucous membrane, which is infiltrated with small cells, tubercular granulations are often seen at the edges of the ulceration, and in the granulating and superficial layer composed of embryonic tissue beneath the ulcerated part, tubercular granulations are also found; and deeper still in the connective tissue which separates the muscular fasciculi of the tongue tubercles are present. All these granulations contain numerous giant cells and obliterated blood vessels. Around them the embryonic tissue is continuous into the interstices of the muscular fasciculi. These morbid changes may be found to extend much further than would be supposed on examining the tubercular ulcer during life. The **uvula** and soft palate may also undergo change in tuberculosis. The uvula is often irregular, inclined to one side, contracted or curved in a permanent manner. Its mucous membrane is sometimes smooth and white, and sometimes irregular from the presence of projecting

tubercular nodules. One or more small tubercular ulcerations may also be seen on it. In these cases we may find chronic

Fig. 82.—Section of a Tubercular Granulation of the Tongue seated deeply between the Muscular Fasciculi.

m, muscular fasciculi ; *t*, tubercular granulation ; *v*, transverse section of the obliterated blood vessels and giant cells ; *n*, connective tissue.

sclerous inflammation of the mucous membrane, inflammatory œdema, or tubercles. The mucous membrane of the uvula is extremely thin normally (*vide* fig. 83). In a section of this organ,

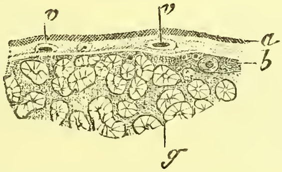


Fig. 83.—Section of the Mucous Membrane of the Uvula in the normal condition.

a, epithelium of the uvula ; *b*, the corium ; *v*, vessels of the mucous membrane ; *g*, glandular culs-de-sac. Magnified 25 diameters.

the mucous membrane is first seen, then the acinous glands which fill up a large part of the section, and at its centre small muscular

fasciculi. When the mucous membrane is chronicly inflamed and sclerosed (fig. 84) it may attain a considerable thickness; it is

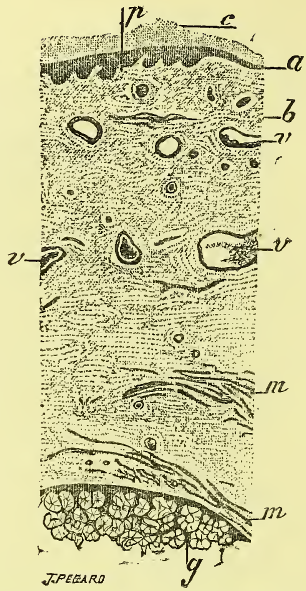


Fig. 84.—Section of a Sclerosed Uvula.

c, superficial layer of the epithelial lining; *a*, the mucosa; *p*, papillary network; *b*, greatly thickened submucous tissue; *v*, much dilated blood vessels; *m*, muscular fibres; *g*, racemose glands. Magnified 25 diameters.

then composed of thick, hyaline bundles of fibrous tissue, separated by beds of flat cells or lymph cells. These blood vessels

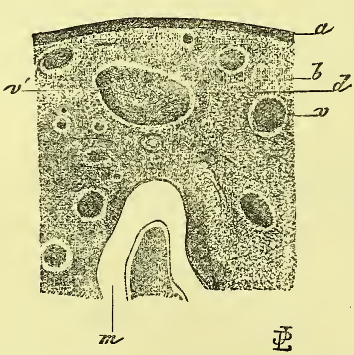


Fig. 85.—Section of the Mucous Membrane of the Uvula in Tuberculosis.

a, epithelium; *b*, connective tissue infiltrated with lymph cells; *m*, a vessel, the contents of which have been removed in preparation; *v*, vessel filled with a fibrinous thrombus; *v′*, larger vessel also filled with fibrin. Magnified 25 diameters.

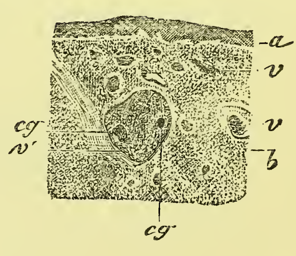


Fig. 86.—Section of the Mucous Membrane of a Tubercular Uvula.

a, epithelium; *b*, mucosa; *v*, vessel; *v′*, vessel containing a fibrinous thrombus, in the midst of which two giant cells, *cg*, are seen. Magnified 25 diameters.

are very numerous and large, though the structure of their walls is that of the capillaries or the small arteries and veins. The papillæ are larger than normally. In a case of subacute tubercu-

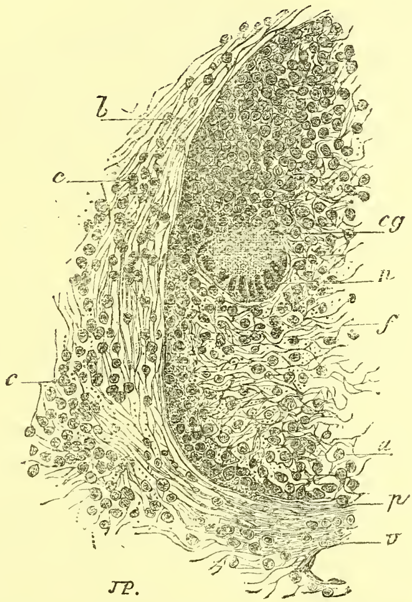


FIG. 87.—SECTION PASSING THROUGH A SEGMENT OF A CAPILLARY GREATLY DISTENDED, AND SHOWING A GIANT CELL INSIDE IT.

cg, giant cell contained in the intravascular fibrinous thrombus; *f*, filaments of fibrin, and *a*, lymph cells of the thrombus; *p*, wall of the vessel; *b*, connective-tissue fibres of the mucous membrane; *c*, *c*, lymph cells contained in the connective tissue. Magnified 250 diameters.

losis of the uvula, which we examined, the mucous membrane was very much thickened and infiltrated with lymph cells; its numerous and large capillaries, newly formed for the most part, were filled by thrombi, the fibrinous meshes of which contained leucocytes and some red blood corpuscles (*vide* fig. 85). This obliteration of the blood vessels existed everywhere throughout the uvula. In sections cut through the granulations very manifest giant cells were often seen, situated in the middle of the vessels, within the intravascular fibrinous thrombus, as may be seen in fig. 87, which, drawn under a high power, can leave no doubt on the subject. Sections passing through small superficial ulcerations of the mucous membrane show crater-like depressions at which the epithelial investment is wanting. These erosions are covered by pus. At the edges of and beneath the ulcerations

numerous tubercular granulations or follicles are present in the thickened mucous membrane. Some of the latter may be recog-

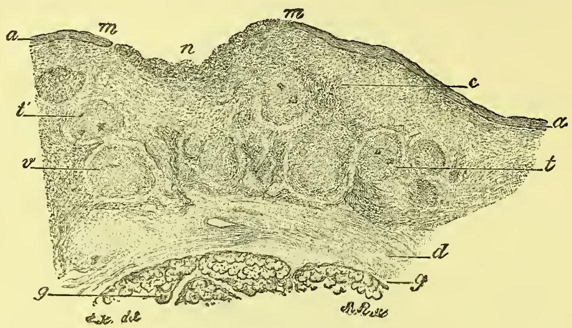


FIG. 88.—SECTION OF A TUBERCULAR ULCERATION OF THE UVULA.

The epithelium, *a*, stops at *m*, *m*, the edges of the ulcer, *n*. At *n* there is a layer of pus. *v*, vessel blocked by a thrombus; *t*, *t'*, similar vessels, each containing two giant cells; *c*, connective tissue infiltrated with lymph cells; *d*, deep part of the mucous membrane; *g*, *g*, racemose glands. Magnified 25 diameters.

nised as nothing else than vessels filled with fibrin in the midst of which giant cells are formed (*vide* figs. 87 and 88).

This description of tubercle of the uvula applies absolutely to tubercular granulations of the soft palate and the back part of the pharynx. **Tuberculosis of the soft palate** causes very marked thickening of the mucous membrane both anteriorly and posteriorly. The submucous connective tissue and that which surrounds the acinous glands is infiltrated with lymph cells and tubercular tissue. The submucous tubercles are often seated at the orifice of a racemose gland, or along the duct. Most frequently the excretory duct of the gland is then dilated and filled with accumulated cylindrical or indifferent cells. The same occurs in the acinous glands of the larynx and trachea, and tubercles and giant cells may even develop in the middle of the glandular acini.

Leprous tubercles are found on all parts of the buccal mucous membrane, on the lips, inside the cheeks, on the tongue, particularly at its base, on the uvula, the soft palate, and the tonsils. They have the same structure as in the skin, where they will be described in detail.

Parasites.—The parasites observed in the buccal mucous membrane are very common and extremely diverse. To examine them it is enough to remove a little of the tartar which accumulates between the teeth, and to mix it with a drop of water on a

slide. Mixed together with saliva corpuscles, a considerable quantity of schizomycetes will be found belonging to the genera bacillus, vibrio, micrococcus, and also ciliated infusoria.

The genus bacillus.—The *leptothrix buccalis* (Robin) exists normally on the papillæ of the tongue. This microscopic alga vegetates on the epithelial cells which form a more or less thick covering to the papillæ. It is also constantly found in the dental tartar, between the large molars, &c. It is characterised by tufts of filaments, which are very fine, rather long, straight or curved. They spring from a granular mass or matrix which is seated either in the modified superficial epithelial cells or on their surface. The filaments of the leptothrix, which are among the largest and longest of the genus bacillus, are formed of segments septate or articulated end to end. They may attain a mean length of 25 μ to 50 μ, but if the tongue and dental tartar be examined in patients who have not taken food for many hours, and before they have washed out the mouth, the filaments may be found from 100 μ to 200 μ in length. They continue to grow after death, so that tufts formed by them may be seen with the naked eye on the tongues of dead persons. In structure the cells forming the filaments are composed of a hyaline membrane, hyaline protoplasm, and the granules described by Robin (1853) as the spores of these algæ. They develop by segmentation of the corpuscles of their matrix or zooglea, or by spores. The *bacillus tremulus* and *bacillus subtilis*, which are also met with in the dental tartar, greatly resemble the leptothrix, but their filaments are shorter and thinner, and their segments are shorter. The rods of the *bacillus tremulus* are animated by movements of translation *en masse*, or by trembling and giratory movements. Their hyaline, mucilaginous envelope is very easily seen. The rods of the *bacillus subtilis* bend and wriggle when moving from spot to spot. These rods are from 10 μ to 16 μ in length and 1 μ in thickness.

The spirochœte denticola (Cohn, Koch) lives not only in the mucus found at the edges of the gums, but also in the dental tartar and on the tongue. The extremely delicate filament of which it is composed is helicoid in form. Its entire length is from 10 μ to 20 μ, and its thickness from 0·2 μ to 0·3 μ. It displaces itself slowly by turning round its longitudinal axis. This spirochœte, which is very similar, if not identical, to the *spirochœte plicatilis*, discovered by Obermeier in the blood of persons affected with recurrent typhus, seems to have some intimate connection in

the mouth with the salivary corpuscles. According to Arndt the spores of the spirochœte develop in the protoplasm of the salivary cells.

The genus vibrio.—The *bacterium termo* is known under two forms, isolated and very mobile, or in zooglea and immobile. They are short oblong cylinders, 2 μ in length and from 0·6 μ to 0·8 μ in thickness, and have a backward and forward movement around their longitudinal axis; this movement is sometimes slow and sometimes so rapid that they dart suddenly out of the field of the microscope. According to Koch, Warning, &c., they have a vibratile cilium. They are often associated in twos, or united into colonies. This microbium is, as we know from the researches of Pasteur, the essential agent in septicæmia. It is always present in the buccal mucus, and it finds a medium favourable for its development in the alimentary detritus which is always present in the mouth. Here also is constantly met a vibrion which, according to Rapin,[1] is related to the *vibrio lineola* or *rugula.* It is from 4 μ to 6 μ in length, and 0·8 μ in thickness; it moves so rapidly, either rotating, turning over and over, or darting along, that it disappears suddenly from the microscopic field of observation. It is composed of small round joints, separated by hardly visible strangulations.

Micrococcus.—In preparations of mucus obtained from scraping the tongue or from the dental tartar, micrococci are always found, either isolated or grouped in colonies. The colonies, or zooglea, are composed of a transparent gelatinous mass sprinkled over with an infinite number of small corpuscles, more or less round in shape, refractile and immobile, and which stain strongly with anilin dyes.[2] These colonies of micrococci are often attached to the epithelial processes of the filiform papillæ of the tongue.

[1] Rapin, *Contribution à l'Etude des Bactéries de la Bouche.* Thèse. Paris, 1881.

[2] To stain bacteria well, methylanilin violet (5 B, the St. Denis make) is used, or 1 per cent. solution of gentian violet (Berlin make), recommended by Weigert. After immersing the dental tartar in a few drops of the solution for about half an hour, the stained fluid is covered with a fine cover glass and examined. To preserve the preparation it may be allowed to dry, or a drop of glycerine, to which carbonate of soda has been previously added, may be introduced under the cover glass; but it is better to extend the dental tartar in a thin layer on the cover glass, to dry in an oven, and stain with the violet. The preparation is washed with distilled water and absolute alcohol, then moistened with oil of cloves, the excess of which is removed by filtering paper; and, finally, mounted in Canada balsam. It is true that by this process a great many of the bacteria are removed, but enough always remain to study. The simpler method may be followed of mixing the tartar with a solution of violet, allowing it to dry, adding a drop of Canada balsam, and covering with a cover glass.

This micrococcus, which might be placed in the group of chromogenic bacteria, plays an important part in the fur of the tongue. It is to it in particular to which Raynaud and Malassez attribute the black fur of the tongue, known under the name of *nigritia.* These black patches on the tongue may be divided into a black central zone and a peripheral zone spotted with black, like a slightly smoked glass. They commence by a black spot which extends and becomes darker. As the patch extends at the periphery the centre desquamates, becomes first red and then normal in appearance. The papillæ of the tongue are at these patches surrounded by myriads of micrococci, which do not differ from those of the normal tongue except by their black colour.

Ciliated infusoria are very common in the mouth. M. Rapin has found the *cercomonas intestinalis* here.

The oïdium albicans.—Of all the parasites of the buccal mucous membrane the most important pathologically is the *oïdium albicans* (Robin), of which the patches of thrush are composed. *Thrush*, which is characterised by small white spots, or soft pulpy patches, is often seen in newborn children, in whom its development is favoured by milk diet. It is generally quite an unimportant accident; but if seen in children and adults in the course of, and particularly in the cachectic period of chronic diseases, it is a serious indication of profound disturbance of nutrition. Its development is in relation to the acid condition of the saliva, an acidity which is itself due to the presence of saccharine or amylaceous substances in a state of fermentation on the surface of the mucous membrane. If a morsel of the white substance of thrush be spread on a slide and examined under the microscope, the different elementary parts of the parasite can be easily recognised. They are: 1st, a mycelium composed of numerous trunks and tubes, which are fistular, partitioned at certain distances, and filled with molecular granules floating in a colourless protoplasm; 2nd, vesicles and spores at the end of the tubes. The spores are oval or round, large, and very numerous.

Cysticerci have in a few rare cases been developed in the lips and in the muscular tissue of the tongue. Two cases are recorded by Dupuytren and Ricord of hydatid cysts, containing echinococci, located in the wall of the mouth.

CHAPTER II.

THE TONSILS.

I. Normal Histology of the Tonsils.

THE tonsils are essentially composed of reticulated connective tissue and lymph follicles. They are covered by the mucous membrane of the pharynx, which dips into the infundibuliform depressions or crypts which open on to their surface, and penetrates also deeply into their tissue. On examining under a low power a section which passes through the long axis of a tonsil perpendicularly to its surface, the mucous membrane will be seen to dip into the depressions and to line their walls. This mucous membrane is lined with stratified pavement epithelium, and is provided with vascular papillæ like that of the mouth ; beneath it are closed follicles scattered through a retiform tissue. Each crypt, lined with its mucous membrane, folded round with adenoid tissue and closed follicles, constitutes a kind of composite follicular gland, surrounded by its own envelope of connective tissue. The organ, considered as a whole, is limited below by a fibrous capsule in which the acini of racemose glands are found as well as large lymph vessels. Normally the crypts of the tonsils contain epithelial cells detatched from the mucous membrane which lines them, and a few round cells resembling lymph cells ; similar cells, called salivary corpuscles, are found on the surface of all parts of the buccal mucous membrane. Micrococci and the filaments and spores of leptothrix are also observed. The tonsils, regular in structure in children, almost always undergo change with age, for there is no organ more frequently affected with primary inflammation or with that secondary to other diseases, e.g. catarrhal inflammation, simple angina, the angina of eruptive fevers, phlegmonous angina, the chronic angina of smokers and drunkards, syphilitic, tubercular tonsilitis, &c. In consequence of these various affections the retiform tissue and the crypts of the tonsil enlarge, become filled with epithelial cells, which, mixed with salivary corpuscles and the filaments and spores of the

leptothrix buccalis, form caseous and fœtid plugs. Thus in the post-mortem examination of adults cavities will almost always be found on dividing the tonsils; they vary in depth and resemble small cysts, but they really communicate with the necks of the crypts and are filled with a caseous detritus. The tonsils are also often atrophied.

II. Pathological Histology of the Tonsils.

Inflammation.—Tonsillar inflammation may be either acute or chronic. Among the former will be included superficial or catarrhal tonsilitis, lacunar tonsilitis, parenchymatous or phlegmonous tonsilitis, tonsilitis which is symptomatic of fevers, and diphtheritic tonsilitis. Chronic tonsilitis includes catarrhal tonsilitis, chronic hypertrophy and atrophy of the tonsils.

Acute catarrhal tonsilitis (*erythematous tonsillar angina, simple angina*).—This consists in superficial redness of the mucous membrane which covers the surface of the tonsils. The mucous membrane of the soft palate and its pillars often shows the same appearances; its capillaries are filled with blood, and its connective tissue is the seat of inflammatory œdema accompanied with extravasation of a few lymph cells which pass through the swollen layers of the epithelium and mix with the desquamated epithelium to form the mucus which covers the morbid parts.

Lacunar catarrhal or cryptic tonsilitis.—Very frequently the mucous membrane which dips into the lacunæ of the tonsil is inflamed simultaneously with that which covers its surface. The whole organ is then swollen and projects outwards between the pillars. The crypts become filled with muco-pus, which oozes from their orifices after the first or second day. These crypts gradually empty themselves by their enlarged orifices, either one by one or simultaneously, and the tonsil rapidly resumes its normal dimensions, the inflammation not lasting more than four or five days. The lesions of the mucous membrane lining the depressions are the same as those of that of the surface, except that the muco-pus secreted in the lacunæ remains imprisoned for a certain time and distends them, thus causing an increase of the total volume of the organ. Sometimes the fluid contained in the lacunæ has the characters of thick pus. On wiping the surface of the tonsils small yellow or grumous spots are found at the orifices

of the crypts; if the pus remains here for a certain time bacteria are formed, and fat, fatty acids, and even cholesterin and calcareous granules.

Parenchymatous or phlegmonous tonsilitis.—Acute parenchymatous tonsilitis is characterised by considerable tumefaction of

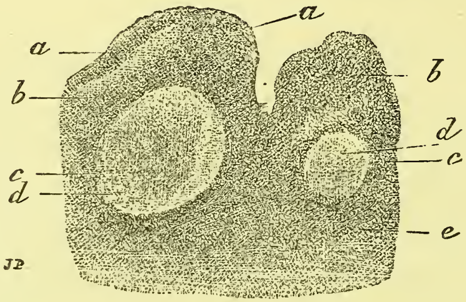


FIG. 89.—SECTION OF THE SUPERFICIAL PART OF A TONSIL IN ACUTE PARENCHYMATOUS ANGINA.

a, a, epithelial lining; *b,* corium infiltrated with lymph cells; *c, c,* retiform tissue of the tonsil equally inflamed; *d, d,* rather large follicles. Magnified 30 diameters.

the whole organ, the retiform and follicular tissue of which is inflamed at the same time as the mucous membrane. In sections of the tonsil it will then be seen that the connective tissue of the superficial mucous membrane as well as that which lines the crypts is crowded with lymph cells; the retiform tissue of the entire gland is similarly affected. The follicles are increased in size by the accumulation of lymph cells, and a similar infiltration is present in the fibrous capsule. The lymph cells are often collected into masses, which form small abscesses. Phlegmonous angina is more particularly that form of angina which terminates in a true abscess, which abscess opens either on the surface of the tonsil or into one of the crypts. The connective tissue behind the tonsil is also sometimes the seat of inflammation which terminates in an abscess.

Tonsilitis symptomatic of febrile diseases.—The angina which accompanies typhoid fever, measles, erysipelas, and variola is generally limited to the superficial mucous membrane of the tonsil, and presents exactly the same characters as in other parts of the buccal mucous membrane (*vide* Stomatitis of Variola, &c., p. 185). Scarlatina often provokes very acute superficial or deep tonsilitis. The inflammation is rarely limited to the surface of the gland, and generally invades the crypts. The

muco-pus secreted on the surface and in the depressions of the gland is thick, spumous, and resembles a soft pulpy false membrane. Sometimes true parenchymatous angina is observed in scarlatina. The tonsil may be even covered with a false membrane absolutely similar to that of diphtheria. In this pseudo-membranous angina of scarlatina the connective tissue of the mucous membrane and the retiform tissue of the tonsil show the same lesions as in diphtheritic angina.

Diphtheritic tonsilitis.—Tonsils affected with diphtheria are generally much swollen, and are covered partly or entirely with thick, felt-like, adherent false membranes, beneath which the mucous membrane is strongly congested and inflamed. The base of the tongue, the soft palate, the uvula, and the pharyngeal mucous membrane are generally affected by the same lesions. On dividing the tonsil it is seen to be of a pinkish grey colour, and it yields a little juice on scraping. The crypt-like depressions which groove it are generally filled by a white or grey opaque exudation of the same structure as the superficial pseudo-membranes. Sometimes the diphtheritic false membranes only occupy a part of the lacunæ, and are spread over their edges and orifices, while the rest of the tonsillar surface is free. In sections of hardened pieces comprehending the whole of the tonsil its increase in size may be estimated, and the extent and thickness of the superficial false membrane may be appreciated, and it will then be seen that the latter is continuous into the lacunæ, and that the follicles of the tonsils are large. The superficial false membrane is composed of layers of fibrin parallel to one another or forming a network. On its surface large balls or masses of micrococci are found united into colonies or zooglea, and small rods. Between the lamellæ and fibrils of fibrin a few lymph cells and red blood corpuscles are found, as well as micrococci, which are free or lodged in the lymph cells, or are gathered into colonies. Beneath the false membrane the mucous surface is generally entirely deprived of its epithelial lining; at the spots where there is no false membrane the inflammatory lesions of the epithelial cells (vesicular, condition, &c.), already described, are met with. The corium, which is in contact with the false membranes, is sometimes smooth, sometimes irregular, and the papillæ and papillary vessels often penetrate into the false membrane which caps them. The connective tissue, all of which is profoundly altered, is infiltrated with lymph cells and red blood corpuscles, and the capillaries contain a great number of leucocytes.

At its maximum this inflammation implicates the whole tonsil. The superficial false membrane is prolonged into the crypts of the tonsil, where, as on the surface, it is usually composed of a network of filaments of fibrin, with lymph cells, epithelial cells, and red blood corpuscles entangled in its meshes. In some cases of diphtheritic tonsilitis the crypts contain no fibrin, but only lymph cells and desquamated epithelium. The epithelial cells are often detached from the papillæ while still adhering together, and they form large plaques, which accumulate in the lacunæ. The papillary network, when peeled off, is seen to be covered by a single layer of cylindrical cells, or simply by migratory lymph cells. Besides the preceding elements the crypts always contain micrococcus spores. The mucous membrane which lines the crypts is as acutely inflamed as that of the surface. The increase in size of the tonsil is not solely due to distension of the lacunæ by exudation; the follicles and the retiform tissue surrounding them also play a great part; they are crowded with lymph cells, and the capillaries, the walls of which have undergone inflammatory changes, contain a large number of lymph cells. In the follicles conglomerations of small atrophied and granular cells are often observed, and between them micrococcus spores are present. The connective tissue surrounding the tonsil is very much inflamed, particularly in the case of primary diphtheritic poisoning of the pharynx. The meshes of the connective tissue are filled with lymph cells, and a network of fibrin is often formed, as in acute phlegmon. The large lymphatic trunks at the base of the tonsil are equally filled with lymph cells. The submaxillary lymph glands are greatly swollen almost from the commencement of the angina. The surface on section is seen to be of a pinkish grey colour, in which opaque granules are visible; these are the hypertrophied follicles. On scraping, a milky juice is obtained which contains spores and small rods. In sections examined under the microscope lymph follicles are observed, containing small granular cells, and micrococcus spores are found accumulated in the follicles and perifollicular sinuses. The blood vessels of the cavernous tissue contain a large number of lymph cells, and their endothelial cells are tumefied.

Chronic tonsilitis.—Chronic catarrhal tonsilitis does not differ from acute tonsilitis except by its duration and its frequent recurrence. Chronic parenchymatous tonsilitis is, on the contrary, a distinct disease, both from its cause, symptoms, and pathology, and merits a special description.

Chronic parenchymatous tonsilitis. Hypertrophy of the tonsils. —Hypertrophy of the tonsils is a disease of infancy, and occurs generally after attacks of catarrhal angina in subjects regarded as lymphatic or scrofulous. The tonsils are very large, hard to the touch, prominent, with a smooth surface, and of a grey or pinkish grey colour. In a section of the large axis of one of these glands, made immediately after ablation, it will be seen that the tissue is grey and slightly vascular. The crypts are reduced to simple slits, the walls of which are in contact; and neither cysts nor irregular depressions, which are so often found in the substance or on the surface of the tonsils in adults, are generally present. On closely examining the section the follicles may generally be seen with the naked eye, with slightly yellow centres,

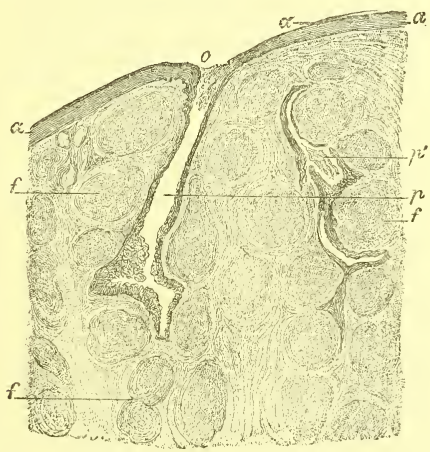


FIG. 90.—SECTION OF AN HYPERTROPHIED TONSIL.

a, *a*, superficial epithelium; *o*, opening of a follicular depression, the cavity of which, *p*, is lined by epithelium continuous with that on the surface; *p'*, cavity of another depression; *f*, *f'*, hypertrophied lymph follicles separated by bands of fibrous tissue. Magnified 20 diameters.

thus resembling the hypertrophied lymph glands of scrofula. In sections made after hardening, and examined with a low power so as to obtain a view of the whole, it will be seen that the crypts are narrow, the lymph follicles large and separated by rather thick bands of fibrous tissue. If in sections treated by a 33 per cent. solution of alcohol the free cells be brushed out of the retiform tissue, the reticulum of the lymph follicles will be clearly seen, as in fig. 91. On comparing these preparations under a low power with those of a normal tonsil the hypertrophy of the follicles will be easily recognised, as well as the thickness of

the fibrous tissue which lines the submucous tissue and separates the follicles. The same preparations examined under high powers show that the epithelium of the surface and the depressions

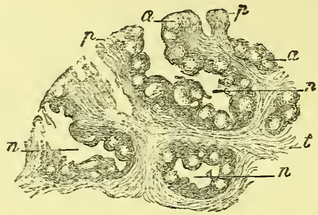


Fig. 91.—Section of an Hypertrophied Tonsil obtained after treating with a 33 per cent. of Alcohol and brushing with a Paint Brush.

a, a, lymph follicles; *n, n,* lacunar depressions; *p, p,* suface of the tonsil; *t,* hypertrophied fibrous tissue. Magnified 6 diameters.

of the tonsils are normal. The crypts contain a few salivary glands and desquamated epithelial cells (fig. 92). The papillæ

Fig. 92.—Elements contained in the Crypts of an Hypertrophied Tonsil.

a, lymph cells or salivary corpuscles; *b,* epithelial cells. Magnified 200 diameters.

of the corium are less developed than normally, and they are even completely atrophied in places. It seems as if the mucous membrane were stretched and pushed back by the hypertrophy of the subjacent tissue. The thickness of the submucous connective tissue and the trabeculæ which separate the follicles is due, not to infiltration by lymph cells, as occurs in acute tonsilitis, described above, but rather to new formation of thick bundles of connective tissue. After staining the sections with carmine these bundles are seen either as stained refractile trabeculæ or circles, according to their section, and separated by connective-tissue cells. The lymph follicles are much altered. Their fibrils have preserved their normal appearance, but the cells entangled in their meshes are larger than normally; their protoplasm is often granular and contains fine fat granules, and their nuclei are oval and large. This state of the cells of the lymph follicles and the sclerosis of the connective tissue makes tonsillar hypertrophy

akin to scrofulous hypertrophy of the lymph glands; but in the former the giant cells are not found which are so numerous in strumous glands. The blood vessels, the small arteries and veins which traverse the fibrous tissue, are themselves highly sclerosed. Their external tissue is much thickened, and their calibre seems to be diminished. Hence the anæmic condition of hypertrophied tonsils and the slight hæmorrhage their removal causes.

Fibrous degeneration of the tonsils.—The tonsils are sometimes observed to be of the ordinary size, or slightly hypertrophied or

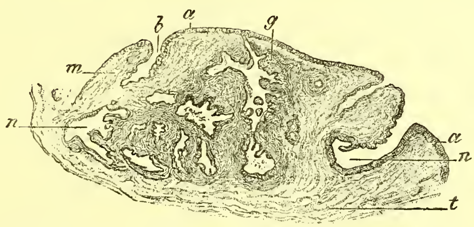


FIG. 93.—SECTION OF A TONSIL WHICH HAS BECOME ENTIRELY FIBROUS.

a, superficial mucous membrane; *b*, crypt; *n*, enlarged depression; *g*, fibrous connective tissue of which the whole tonsil is formed; *t*, connective tissue at the base of the tonsil. Magnified 5 diameters.

atrophied, but very hard, and the crypts enlarged. On cutting

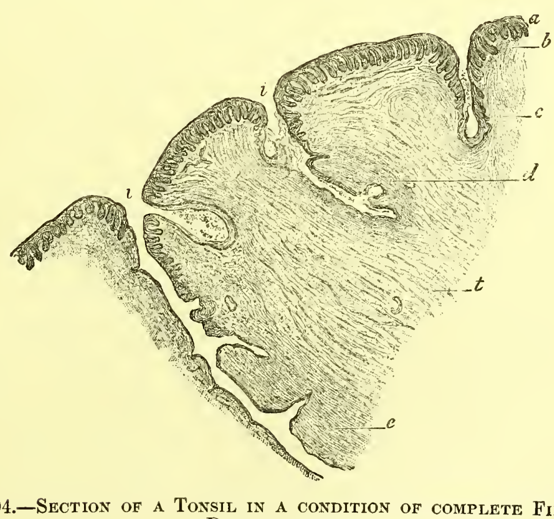


FIG. 94.—SECTION OF A TONSIL IN A CONDITION OF COMPLETE FIBROID DEGENERATION.

a, superficial epithelial investment; *b*, papillary network; *c*, *d*, fibrous submucous tissue; *t*, fibrous tissue at the centre of the tonsil; *i*, *i*, orifice of the crypts. Magnified 10 diameters.

them across the crypts are found to be so large that the tonsil

seems to be formed of isolated buds; sometimes cystic cavities are found at their bases filled with caseous matter. In sections made after hardening, and examined under a low power, the lymph follicles are found to be either partly or entirely absent, and the whole tissue of the tonsil to be fibrous. Under a high power the papillæ of the mucous membrane are seen to be greatly developed, and the submucous tissue extremely dense and composed of fibrils which are finer and closer together than at the centre of the tonsil. The blood vessels have thick sclerous walls. This lesion is observed in adults as the result of repeated angina. We have frequently seen it in subjects who have died from pulmonary phthisis when there are no tubercles in the tonsil itself. In some subjects a sclerosed tonsil is much atrophied. Only the small orifices of the crypts can be recognised and the organ is composed of a hard and flattened plaque.

Syphilitic lesions of the tonsil.—Mucous plaques of the tonsil, which may often be studied in tonsils removed during life, are formed, as in every other part of the mucous membrane, by hypertrophy of the infiltrated papillæ and the presence of numerous lymph cells in the mucous corium. The deep and superficial layers of the epithelium are equally thickened and infiltrated, and crowded with migratory lymph cells. These are accumulated on the surface of the epithelial investment between the flat cells, or are more deeply placed between the polygonal cells of the Malpighian layer; they form small microscopic abscesses, which open on the surface.[1] The brilliant white surface of these plaques is owing to lesions of the epithelium, and their prominence is caused by the simultaneous inflammatory thickening of the epithelium and the mucous corium. In fact, mucous plaques do not only implicate the superficial mucous membrane of the tonsil, but they cause similar lesions of the mucous membrane lining the crypts, whence follicular catarrh of these organs results. Finally, the reticulated tissue of the follicles becomes itself chronically inflamed and parenchymatous tonsilitis is produced, even if the disease is not limited to the tonsils, and more or less chronic hypertrophy similar to the syphilitic lesions of the lymph glands already described (*vide* vol. i. p. 558). Mucous plaques of the tonsil rarely ulcerate; the lymph cells and blood corpuscles which infiltrate the epithelial investment pass through the layers, and are eliminated without destroying them; but a true erosion or ulceration may be produced superficially, and may be even covered

[1] *Leçons sur la Syphilis*, Cornil (1879), p. 118.

with a fibrinous pseudo-membrane when the inflammation is very acute. Tertiary syphilis shows itself in the tonsils by gummata followed by deep ulcerations.

Tubercle of the tonsils.—Much light has been recently thrown on tuberculosis of the tonsils by the researches of Isambert, Peter, Laboulbène, Barth, Chassagnette, &c. It is seen in the acute state in the form of miliary granulations, or in the chronic state in the form of more or less deep caseous ulceration. Miliary tubercles of the tonsil are composed of small nodules which are developed either in the superficial layer of the mucous corium or in the reticulated follicular tissue; they are at first grey and semitransparent. They coincide with rapid general tuberculosis. More frequently, however, tubercle of the tonsil is chronic, and is developed in an isolated manner in the mucous membrane which covers it and in its parenchyma. The tubercles soon become yellow and caseous at their centre. Those which are situated in the superficial mucous membrane show an opaque yellowish projection, which soon undergoes ulceration, and a small erosion is formed with a yellow base, which is filled with a grumous fluid. The tubercles which are deeply seated implicate a certain number of the follicles and are accompanied with more or less extensive tubercular infiltration. On post-mortem examination of tonsils affected with marked tuberculosis a few prominent tubercular granulations or erosions will first be observed on the surface. The necks of the crypts are enlarged and exude a thick caseous pus. On dividing the tonsil the crypts are generally seen to be much enlarged, filled with a caseous detritus, and bordered by a tissue in which yellow and opaque spots are seen; these are the centres of tubercles. On examining sections of such a tonsil under the microscope diffused tubercular infiltration, or granulations developed in the mucous corium, will be found under the epithelium and at the ulcerated points not covered by epithelial tissue. This newly-formed tissue is scattered over with giant cells. If the section passes through a crypt (*vide* fig. 95) it will be seen that the cavity is at first enlarged and filled with epithelial cells and pus cells. The epithelium of its wall is more or less detached, and the papillæ are generally hypertrophied and budding. The proper tissue of the tonsil, that is to say, the connective tissue and the follicles situated around a crypt, is changed into a tubercular tissue, the granulations of which are severally well marked, and in their centre giant cells are found (c, c', c'' fig. 95). Lymph follicles are also frequently found

which have preserved their form and structure, but in which tubercles with giant cells are developed. The tubercular ulcera-

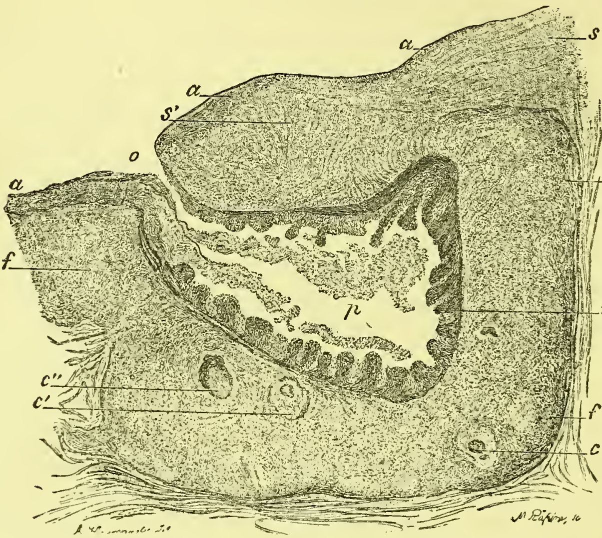


FIG. 95.—SECTION OF A TUBERCULAR TONSIL.

o, orifice; *p*, cavity of the crypt containing a caseous detritus; *m*, much-developed papillæ belonging to the mucous membrane lining the cavity; *a*, *a*, mucous surface of the tonsil; *s*, its thickened connective tissue; *f*, *f*, follicles; *c*, *c'*, *c''*, centres of tubercular granulations with giant cells. Magnified 30 diameters.

tions on the surface of the tonsil are edged, as all ulcerations of the same kind, by tubercular tissue and granulations with giant cells; ulcerations of the same kind are developed on the surface and at the bottom of the crypts; whence result large anfractuous ulcerations on the surface of the organ, separated by buds of tonsillar connective tissue, which is still intact between the crypts. All this newly-formed tissue mortifies and is gradually eliminated. At the end of this series of changes the tonsil is sometimes reduced to a small mass of tubercular tissue. In these cases the connective tissue, which forms the deep envelope of the organ, and the adjacent muscles are infiltrated with lymph cells and tubercular granulations, and the pharynx, the base of the tongue, the uvula, and the soft palate are more or less invaded by tubercles. The blood and lymph vessels show the lesions which are always observed in every organ affected with tuberculosis.

Tumours such as lymphatomata, carcinomata, and epitheliomata are rarely observed in the tonsils; if present they show the

same general characters as in the lymph glands. Epithelioma and carcinoma are sometimes developed primarily.

Calculi, composed essentially of phosphate and carbonate of chalk, are sometimes produced in the crypts of the tonsil.

Parasites.—Echinococcus cysts have been observed in the tonsil, but the parasites most frequently met with are those which vegetate on the mucous membrane. Thrush (*oidium albicans*) is developed here as on the rest of the mucous membrane, and is accompanied with superficial inflammation of the tonsil and its crypts. Bacteria and other microbes of the mouth often penetrate into the tonsillar crypts.

CHAPTER III.

THE SALIVARY GLANDS.

I. Normal Histology of the Salivary Glands.

The salivary glands, properly so called, that is to say, the parotid, the submaxillary, and the sublingual glands, are acinous glands. In the sublingual all the saccules contain mucous cells, in the submaxillary gland only a few of the saccules contain them, and in the parotid gland they are altogether absent. The saccules of the latter gland contain finely granular cells, and similar cells are observed in the midst of the acini of the submaxillary gland. The other acini of this gland and all those of the sublingual gland contain two species of cells; mucous cells, which are large, clear, refractile, and pyramidal, with their apex turned towards the centre of the saccule. At the base the nucleus is found in a small protoplasmic process of conical form; it is irregular in form, varying with the pressure of the mucous mass accumulated in the cell. It is to this mass of mucus that the peculiar aspect of these cells is due. On examining them in good sections, made after the action of alcohol and stained with picrocarminate of ammonia, a slightly stained yellow reticulum will be recognised in their interior, formed of fine granular trabeculæ, unequal in thickness, which extend from the mucous globe into the protoplasm which is at the periphery of the cell or accumulated around its nucleus. From this arrangement it results that the mucous substance elaborated by the cell is formed in the midst of its protoplasm in a series of distinct apartments, and that the small drops of this substance, whilst increasing in volume and showing a tendency to fuse together, remain nevertheless separated from one another by delicate protoplasmic layers, which probably still take part in their growth. The second species of cells observed in the acini containing mucous cells differ totally from the preceding. They are small, granular, irregular, and are united in small groups which, occupying the base of the glandular saccules and being pressed by the mass of the mucous cells against the

surface of the glandular membrane, are seen in section in the form of crescents (crescents of Gianuzzi). These cells, which are intimately united, have a convex external surface and a concave internal surface, into which is fitted the outer part of a mucous cell; crests are often observed which are insinuated between two mucous cells. The membrana propria of the glandular

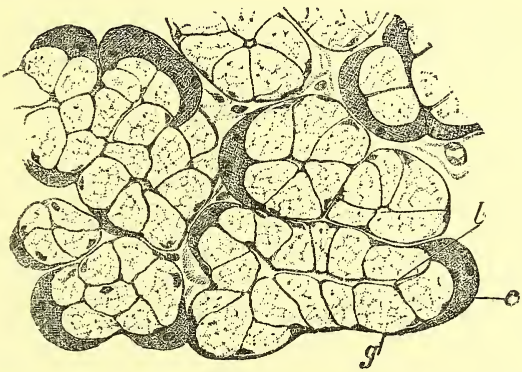


FIG. 96.—SECTION OF A NORMAL SUBMAXILLARY GLAND OF A DOG, MADE AFTER BEING HARDENED IN ALCOHOL, STAINED WITH PICROCARMINATE, AND MOUNTED IN GLYCERIN.

g, mucous cells; *c*, crescent of Gianuzzi; *l*, lumen of the glandular saccules.

saccules, whether they contain mucous cells or not, are not composed of lamellæ, but, as Boll has well established, of flat cells with crested processes united together. The different saccules have spaces between them in which the lymphatic plasma circulates, which is elaborated by the capillaries. All the glandular ducts, whether of the parotid, submaxillary, or sublingual glands, have a single layer of cylindrical epithelium. In the smallest ducts which communicate directly with the saccules the cylindrical cells are short, so that the name of cylindrical is hardly applicable to them; but in the small ducts, which result from the union of many ducts, the cells are distinctly cylindrical and are interesting in structure. The portion between the nucleus and the base of implantation of the cell shows very irregular striæ parallel to its axis (Henle). Pflüger thought that these striæ were produced by terminal nervous filaments; but it is much more probable, as we long ago remarked, that this arrangement indicates the presence of contractile parts which take an active part in excretion; this is important, as in all the ducts of the salivary glands, from the smallest up to the ducts of Wharton and Stenon, there is not a single muscular fibre.

II. Normal and Pathological Physiology of the Salivary Glands.

The mechanism of the secretion of the salivary glands and the modifications which take place in these glands, when in a condition of activity, are two questions intimately united and of a very great importance. Heidenhain was the first physiologist who made a histological analysis of a mucous gland after it had been made to secrete abundantly by exciting the nerves which supply it. He thought thus to exactly determine the mechanism of secretion; though his conclusions were wrong his efforts are not the less meritorious. After exciting, for many hours, in a dog, by means of an interrupted induced current, the chorda tympani, which is the chief nerve of secretion of the submaxillary gland, he thought he recognised in sections of this gland, that all the mucous cells had been thrown off to form the product of secretion, and that they had been replaced in the interior of the glandular saccules by the hypertrophied and proliferating crescentic cells of Gianuzzi. But this is not the case, as we pointed out long ago.[1] If in a dog a tube be fixed in Wharton's duct so as to collect the saliva, and if the chorda tympani be exposed for four or five hours, almost a hundred grammes of saliva will be collected, and the corresponding submaxillary gland

Fig. 97.—Section of the Submaxillary Gland of a Dog, after an abundant secretion for four hours had been produced by exciting the Chorda Tympani (the same preparation and the same power as in fig. 96).

g, glandular cells from which the mucus has been expelled by secretion and the nuclei increased in size; *c*, crescent cells of Gianuzzi which have undergone marked increase in size.

will be found to have undergone considerable changes, which may be studied by comparing the gland with that of the other side,

[1] *Vide* first French edition of Frey (1871), note, p. 437.

which has not been subjected to irritation. To obtain good preparations of such a gland various methods are used. The most simple consists in hardening portions in alcohol, after which extremely delicate sections can be cut, stained with picrocarminate, and preserved in glycerin; the glands may also be hardened in bichromate of ammonia and stained with purpurin; or osmic acid may be used as the hardening fluid, if very small portions be taken. In these preparations it may be seen that the mucous cells are not expelled *en masse* to form the secretion, as Heidenhain said, but that the mucus elaborated in their interior is alone eliminated; at the same time the protoplasm around the nucleus swells, increases in quantity, and is pushed into the interior of the cell to replace the mucus in proportion as this is eliminated. In the same preparation the different phases of this process may be observed, for all the acini are not equally modified; there are some which still appear intact, while others have undergone complete transformation. In the latter all the cells, the mucous cells, and the crescent cells, show the same characters, and

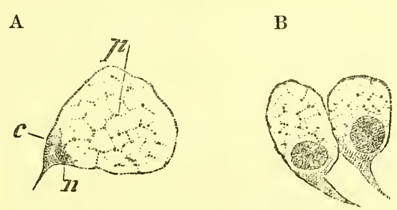


Fig. 98.—A, Isolated Mucous Cells of the Normal Submaxillary Gland of the Dog, after the action of a 33 per cent. solution of Alcohol and stained by Picrocarminate.

B, Cells of the Submaxillary Gland of the Dog prepared by the same method after active secretion of the gland by irritation of the Chorda Tympani.

if the observation be not extended to neighbouring acini, in which the mucous cells still show various-sized masses of mucus at their free borders, one might be led to admit the interpretation of Heidenhain.

The saliva obtained by exciting the chorda tympani is mucous and stringy, but much less thick than that obtained after exciting the sympathetic filaments distributed to the gland; the secretion is still thinner if morphia has been given the dog before irritating the chorda tympani. The saliva of a dog under the influence of morphia shows, under the microscope, a great number of bodies which are as transparent as glass, and slightly more refractile than the fluid in which they float. They vary in form; they may be

cylindrical with blunt extremities, or pyriform, fusiform, and sometimes almost spherical. These are salivary moulds formed entirely of mucous globes; they were mistaken by Heidenhain, who used morphia in his experiments, for mucous cells expelled entire from the glandular acini. A certain number of very characteristic lymph cells are also met with in the saliva, whether it be obtained by exciting the chorda tympani with or without morphia, or by exciting the sympathetic. These cells are probably introduced into the excretory ducts by diapedesis, which is stimulated by the dilatation of the blood vessels, caused by irritation of the chorda tympani. This fact was discovered by Claude Bernard. All the white cells which escape from the vessels in consequence of the vaso-dilating action of the chorda tympani do not necessarily pass into the saccules and glandular ducts; the majority of them are arrested in the interacinous spaces or in the meshes of the connective tissue between the glandular acini, and are taken up later into the lymphatic circulation. By a very simple experiment diapedesis, and the subsequent accumulation of white cells, may be increased. If, as was done by Ludwig, who discovered the physiological action of the secretory nerves, a tube be introduced into Wharton's duct, and put into communication with a mercurial manometer, and if the chorda tympani be excited, the gland will then secrete under a pressure of twenty-five centimetres of mercury; it will then be seen to swell and form a marked projection on the side of the neck. This increase in size is not solely due to the fact that the product of secretion is accumulated in the acini, but to active œdema produced in the interacinous spaces and connective tissue. This œdema, the mechanism of which is now perfectly understood, depends on the fact that tension of the blood in the capillary vessels is increased by the dilating action of the chorda tympani, and by the simultaneous pressure produced on the small veins by the acini, slightly swollen by retention of their secretion. This is an experiment similar to that of tying the inferior vena cava and of cutting the sciatic nerve to induce œdema of the corresponding lower limb. Active œdema of the submaxillary gland is accompanied, as we have already shown, by marked diapedesis of leucocytes, which fact may be recognised by removing fragments of the œdematous connective tissue with the scissors and spreading them on a slide for microscopical examination. This experiment is interesting in that it explains certain pathological phenomena occurring in the salivary glands, particularly in the

parotid in the complaint known as **mumps.** This affection, characterised by considerable and rapid swelling, can only be explained by vasomotor phenomena similar to that experimentally produced in the submaxillary gland of the dog. But, as mumps are contagious, it is probable that the irritation, which causes this active œdema of the parotid gland, is a parasitic microbe. This microbe, which has been observed in the blood of patients affected with mumps, and has been cultivated by M. Pasteur, penetrates, probably by way of Stenon's duct, vegetates in the ducts and saccules of the gland, and excites by its presence either the terminal nerve filaments or the numerous ganglionic cells contained in the thickness of the gland; it then multiplies in the blood, producing a general febrile condition.

If, instead of studying what takes place in glands when excited to secrete abundantly, the changes are observed which occur when their functions are entirely suppressed, as, for example, after ligature of Wharton's duct, it will be seen that, instead of swelling and becoming hypertrophied in consequence of the accumulation of secreted products and secretory elements, they finally undergo considerable atrophy. In sections of an atrophied gland the atrophy will be seen to have affected the glandular elements, while the connective-tissue parts of the organ are hypertrophied; in fact, true cirrhosis is produced. On examining the saccules and glandular ducts more closely, one is struck by the fact that all the cells lining them have become similar; the mucous cells, the crescent cells of Gianuzzi, the short cylindrical cells of the smallest ducts, and the striated cylindrical cells of the principal ducts, have all taken one form, that of embryonic glandular cells; that is to say, they are all similar to cells observed in the gland during development. Ten months after ligature of Wharton's duct the glandular saccules are not distended by accumulation of the products of secretion, and in the principal ducts moulds are found in the form of refractile, yellowish masses, formed of a substance more colloid than mucous in character. Perhaps they result from transformation of the mucous substance first accumulated in the excretory ducts. The important fact learnt from this experiment is that the function of a gland being abolished, the differentiation formerly present in its epithelial elements, which was necessary for its function, completely disappears, all the cells returning to the embryonic or indifferent type. This fact ought not to surprise those who remember how the mamma, after undergoing considerable changes in its glandular elements during lactation, returns

to its primitive condition when its transitory functions have ceased.

FIG. 99.—SUBMAXILLARY GLAND OF A DOG TEN MONTHS AFTER LIGATURE OF ITS EXCRETORY DUCT. SECTION MADE AFTER HARDENING IN ALCOHOL, STAINED IN PICROCARMINATE, AND PRESERVED IN GLYCERIN.

a, glandular succules; *t*, excretory duct; *u*, arteriole.

The different experiments on the submaxillary gland of the dog passed in review may serve to elucidate a series of morbid processes which take place in the mucous glands of the mouth and in the glands generally. When, in consequence of inflammation or some neoplasm, the function of a gland is suppressed for a long time, the glandular elements cease to be differentiated, and they gradually return to the embryonic form, which they retain as long as the function remains suppressed. They may even give origin, as may be seen in the mamma, and particularly in the parotid gland, to active new growths in the form of epithelial buds pushed out in different directions. If by chance the function of the gland is re-established, these elements may, in consequence of a new differentiation, be physiologically restored in the adult. But if the function of the gland is suppressed definitely, as may occur from obliteration of its excretory duct, it undergoes more or less marked atrophy, and connective-tissue elements partly fill up the spaces left by the atrophied and lost elements. This is what occurs in cirrhosis of the liver, cirrhosis

of the kidney, and other glandular organs. This is one of the causes of cirrhosis, for this change follows a series of other influences affecting the connective tissue of the viscera.

III. Pathological Histology of the Salivary Glands.

Inflammation of the salivary glands, catarrhal parotiditis, mumps.—The name of mumps is given to a febrile disease which is particularly common in children; it is generally epidemic, and is of a special and probably of a zymotic nature. It is characterised by swelling of the parotid gland, and sometimes by metastasis in the breasts or testicles. The tumefied parotid glands never suppurate, and this fact distinguishes mumps from phlegmonous parotiditis. As this disease is not fatal, autopsies, in which the anatomical condition of the gland can be observed, are very rare. According to Virchow the glandular lobules of the parotid are prominent and congested, and their ducts filled with muco-pus; the periglandular connective tissue is also congested and infiltrated by serum.

Phlegmonous parotiditis.—Acute inflammation of the parotid, with tendency to end in suppuration, varies according to its cause. It results, in fact, either from irritation conveyed by means of Stenon's duct (stomatitis, mercurial stomatitis, retention of the saliva by a calculus or tubular inflammation of Stenon's duct) or from inflammation of a neighbouring organ, propagated by the cellular tissue of the parotid sheath (adenitis, anthrax, arthritis of the temporo-maxillary articulation). Finally, metastatic phlegmonous parotiditis may be observed in purulent inflammation, puerperal pyemia, acute pneumonia, typhoid fever, cholera, dysentery, variola, glanders, diphtheria, &c. In these conditions its progress is extremely rapid and the prognosis grave. In the pneumonia of old subjects, for example, it appears suddenly and the gland is from the beginning infiltrated with pus. After death a parotid gland affected with phlegmonous inflammation is seen to be much hypertrophied; the connective tissue surrounding it is œdematous, or even infiltrated with a cloudy or puriform fluid, and Stenon's duct is filled with muco-pus. On cutting across the gland the glandular acini and lobules are seen very clearly and are larger than normally. At the commencement of the phlegmonous inflammation the glandular lobules are not all affected to the same degree; some, in fact, are of a red or dull red colour, and others yellowish or opaque. The trabeculæ of connective tissue

which separate the acini and lobules are thickened and infiltrated with serum. At all parts of the gland where the inflammation is of rather long standing a small drop of pus is seen in the centre of the acini. When the parotid has been affected throughout its whole extent by very rapid suppuration, all the lobules and acini are seen on section to be of a yellow colour, and on passing the scalpel over them a cloudy fluid is obtained, having all the characteristics of pus. At the commencement of parotiditis the cells of the acini are seen on microscopical examination to have become granular, in the same way as the cells of the acinous glands of the trachea (*vide* p. 18); lymph cells are, moreover, accumulated in the interior of the saccules; at the same time the septa of connective tissue, which separate the saccules and glandular lobules, are infiltrated with lymph cells. The cylindrical cells of the glandular ducts are undergoing proliferation, and are separated by lymph cells. All the constituent parts of the gland are inflamed simultaneously, and the congestion is sufficiently acute to produce diapedesis of the red blood corpuscles into the connective tissue and glandular saccules. In the acini, which have become yellow and opaque, great numbers of lymph cells fill the saccules, the glandular ducts, and the connective tissue. Pus collects rapidly, either in the acini or in the periglandular cellular tissue. The tumefied gland is at first bound down by its aponeurosis; but the pus which fills the parotid sheath finally makes a passage across the neighbouring parts to the skin. If the inflammation is very rapid and acute, the inflamed parotid, compressed by its aponeurosis, may be partly sphacelated. This gangrenous focus may open in the skin or into the pharynx, or into both at the same time; sometimes hæmorrhage may occur from gangrenous ulceration of the wall of a vessel. Large branches of the external carotid and the jugular vein have been thus compromised, and phlebitis affecting the jugular vein and the cavernous sinuses has been observed as a complication of abscess of the parotid. Hitherto we have been considering serious forms of phlegmonous parotiditis implicating the whole gland, but parotiditis may be harmless if the gland be only partially affected. In one of these forms of chronic and benign parotiditis the gland is partially tumefied, and pus may be squeezed out on pressing Stenon's duct; the inflammation then seems to be limited to the duct or to a few ducts leading to a group of acini.

Simple hypertrophy, or adenoma.—Formerly Bérard, Bauchet,

&c., described as simple hypertrophy, or adenoma, many tumours of the parotid which are now classed among pseudo-carcinomata and chondromata. Since tumours have been subjected to microscopical examination adenoma of the salivary glands has been found to be extremely rare. The title of adenoma of the parotid should be limited to tumours characterised by simple hypertrophy, that is to say, hypertrophy in which the normal histological type of the gland is preserved (*vide* vol. i. p. 284).

Salivary cysts.—There are a few recorded cases of salivary cysts or sacs, varying in size from that of a fowl's egg to a fist, which have been seated in the parotid. These cysts refill after emptying, and they swell during mastication ; it is hence probable that they communicate with the excretory ducts of the gland, but we do not know the exact anatomical relation.

Salivary calculi.—Salivary calculi are sometimes found in the condition of sand in salivary sacs. When located in the excretory ducts of the glands they cause more or less considerable dilatation of the ducts. They have been particularly observed in Wharton's duct, but they are also sometimes seen in Stenon's duct and in that of the sublingual gland. When a single calculus occupies the interior of a salivary duct its form is ovoid with its long axis parallel to the duct; it may attain one to two centimetres in length; it sometimes presents one or more longitudinal grooves past which the saliva continues to flow. It is formed of concentric layers, and is composed in a great part of phosphate and carbonate of lime, with from 7 to 25 per cent. of organic matter. Behind the calculus an accumulation of saliva is found in the dilated duct. The irritation which it produces causes thickening of the duct and a purulent secretion, which mixes with the saliva. Sometimes the wall of the duct ulcerates, and the calculus passes into the neighbouring connective tissue, where it remains encysted. The submaxillary gland has been seen to inflame and to become indurated in consequence of a calculus in Wharton's duct.

Sarcoma.—The different forms of sarcoma, encephaloid, fasciculated, cystic, &c., may be observed in the parotid gland. At the same time that the inter-acinous connective tissue gives origin to sarcomatous tissue the epithelium of the gland returns to the embryonic condition, proliferates, and forms epithelial buds, which are pushed out in various directions. Sometimes also cysts, varying in size, are developed, the walls of which are lined with epithelial cells; they contain a fluid which is generally

milky in appearance from the presence of cellular elements. Finally, it is not rare to meet with tumours in the parotid, which, though distinctly sarcomatous throughout the chief part of their mass, contain islets of mucous tissue and nodules of cartilage. In these cases it is difficult to designate the tumour ; but if one considers that the course, and consequently the gravity, of the disease is dominated by the presence of sarcomatous tissue, one does not hesitate to give the morbid tissue the name of sarcoma, rather than that of chondroma or myxoma, or at least we may imitate Virchow and call it chondro-myxo-sarcoma. We ourselves see no objection, but we may remark that, to be logical, the name of the tumour should be more complicated ; in fact, as it contains glandular epithelium and newly-formed blood vessels, it should be called epithelio-angio-chondro-myxo-sarcoma.

Myxoma.—But few tumours of the parotid are composed solely of myxomatous tissue. Mucous tissue is, on the contrary, frequently found mixed with cartilaginous tissue in chondromata of this gland. In pure myxoma of the parotid the mucous tissue surrounds the saccules of a more or less considerable portion of the gland, or it forms masses developed in the large fibrous septa, in which glandular saccules are no longer found. The structure of these tumours resembles that of ordinary myxoma (*vide* vol. i. p. 154).

Carcinoma.—Carcinoma of the parotid is much rarer than sarcoma; scirrhous and encephaloid carcinoma have, however, been observed. These tumours, scarcely separable from the neighbouring tissues, have no connective-tissue envelope; after perforating the aponeuroses they reach the skin and form buds which adhere intimately to the thickened derm. On dividing and scraping them a milky juice is obtained. Sometimes the lobules of fat which belong to the subcutaneous or intralobular connective tissue of the gland are preserved. The facial nerve and the blood vessels of the region are more or less compromised and blended with the morbid mass. Histological examination reveals the characteristic stroma and cells of carcinoma.

Epithelioma.—Many cases of epithelioma of the parotid, observed by Robin, Verneuil, Billroth and Rindfleisch, correspond entirely to the description of tubular epithelium. These tumours are ill-defined, without a fibrous envelope ; they are grey or yellowish grey in colour, and are composed of ramifying and anastomosing epithelial cylinders. Cell-nests may be met with in the cylinders formed of pavement epithelium.

Chondroma of the salivary glands.—Chondroma of the parotid is rather common; the submaxillary and the sublingual glands are more rarely affected. Since the 'Memoirs' of J. Müller, chondroma of the parotid has been well studied by Dolbeau, Virchow, &c. Two anatomical varieties may be distinguished: pure chondroma, in which the morbid mass is entirely composed of cartilage; and mixed chondroma, in which cartilaginous fibres and mucous tissue may be found at the same time as modified glandular saccules, which might be referred to adenoma; but we think that these complex tumours may be classified among chondromata. We know, in fact, that in every chondroma fibrous tissue is met with and islets of embryonic tissue, and that the glands, whatever may be the primary tumour affecting them, always show irritative lesions and various changes in their saccules. Pure chondromata of the parotid are tumours hemispherical in shape, smooth or lobulated, and about the size of a hen's egg, and are separated from the neighbouring parts by a fibrous envelope. On cutting across the tumour the physiological characters of hyaline cartilage are recognised, either throughout its whole mass or in nodules separated from one another by fibrous tissue. All the structural varieties of cartilage already described, when considering chondroma generally (*vide* vol. i. p. 219), are met with in the salivary glands. The morbid tissue undergoes various changes; cysts filled with a mucous or blood-stained fluid may be met with, or mucous tissue, or masses of cartilage infiltrated with calcareous salts, or even osseous tissue. In most chondromata of the parotid the glandular acini are modified; they may be atrophied, choked by the morbid tissue developed around them; sometimes they are dilated and cystic, but most frequently the cells composing them multiply and form epithelial buds thrown out in various directions.

Chondromata of the salivary glands always show in their mass various tissues derived from the proper elements of the gland; but these must be distinguished from mixed chondromata, in which either a large quantity of mucous tissue is found, or entire lobes showing the structure of myxoma, or a considerable mass of embryonic tissue around cartilaginous lobules. Chondroma of the salivary glands is generally developed from the connective tissue which separates the acini. In a research, carried on under the direction of Recklinghausen, Wartmann found, in two chondromata of the parotid, tubes with delicate walls, anastomosing together and containing epithelial cells; he considered them to

be lymphatics filled with proliferating endothelial cells. These cells may completely fill the lumen of the lymph vessels, so that they appear in transverse sections as small epidermic pearls or globes, and in longitudinal sections as epithelial cylinders. These cells of the lymph vessels are distributed in the neighbouring connective tissue, and become the points of departure of new formation of cartilage by surrounding a capsule, while the fibrous tissue becomes hyaline, cartilaginous, or mucous. The newly-formed cartilage cells contain glycogen. Chondromata of the salivary glands are generally benign tumours, and do not return after removal. Cases have, however, been recorded of relapse; one was published by Wartmann in which the tumour returned in situ, and in the pinna of the ear, and occasioned numerous cartilaginous emboli in the arterioles. In sections of arterioles from $\frac{1}{2}$ to 1 mm. in diameter the lumen of the vessel was seen to be occupied by a collection of cells, the most central of which were surrounded by capsules and hyaline intercellular substance. At the periphery of the cartilaginous thrombi the cells were flattened, imbricated, in contact with one another, and seemed as if they had been derived from the proliferating cells of the tunica interna. In consequence of these vascular changes cells which are centres of cartilage formation spread into the neighbouring connective tissue and give origin to new cartilaginous tissue. According to these observations chondroma may be developed from the epithelial cells of lymph- and blood vessels as well as from connective-tissue cells.

CHAPTER IV.

THE PHARYNX AND ŒSOPHAGUS.

I. Normal Histology of the Pharynx and Œsophagus.

The walls of the pharynx and œsophagus are each composed of four layers: 1. A peripheral aponeurotic envelope composed of bundles of connective tissue and elastic fibres. 2. A thick muscular layer composed in the pharynx of the levator and constrictor muscles, all striated, in the œsophagus of two layers, the external, formed of longitudinal fibres, and the internal of circular fibres. Some of the longitudinal fibres of the œsophagus emanate from the inferior constrictor; others are inserted into the cricoid cartilage; they are striated in the neck. In the thorax smooth muscle fasciculi are added first to the annular fibres, then to the longitudinal; they become more and more numerous as the striated muscle fibres disappear, none of which are present in the lower third of the œsophagus. The longitudinal bundles of the œsophagus send expansions to the posterior part of the trachea, to the left bronchus, &c. 3. A layer of submucous connective tissue. 4. A mucous membrane, the structure of which varies according to the parts examined.

The mucous membrane of the pharynx is divided into two very different regions. Below the posterior pillar of the soft palate it is lined, like that of the mouth, with pavement epithelium, and it possesses rudimentary papillæ. At the upper part, on the contrary, that is to say, on the posterior surface of the uvula, and on the soft palate, around the orifices of the Eustachian tubes, at the posterior opening of the nasal fossæ, and on the roof of the pharynx, it is lined with ciliated cylindrical epithelium; it has no papillæ and it contains a large number of glands. The mucous membrane of the œsophagus is invested, like that of the lower part of the pharynx, with a stratified pavement epithelium; it is furnished with conical papillæ, which are much more developed than those of the pharyngeal mucous membrane, and in its deep layer it contains smooth muscle fibres. Throughout the whole

extent of the pharyngo-œsophageal mucous membrane there is a rather considerable number of racemose glands. In the pharyngeal mucous membrane, moreover, simple lymph follicles are met with, as well as composite follicles, such as those of the tonsils. These composite follicles form a continuous layer between the orifices of the Eustachian tubes many millimetres in thickness. They are also rather numerous at the middle part of the roof of the pharynx, near the posterior nares, on the posterior surface of the soft palate, and in the walls of the pharynx as far as the epiglottis. The racemose glands, which are easily recognised with the naked eye, by the relief they form under the mucous membrane and by the orifices of their excretory ducts, are also located in the upper part of the pharynx, on the posterior surface of the soft palate, around the openings of the Eustachian tubes, &c. They are particularly numerous on the whole of posterior wall of the pharynx, and they become rarer as the œsophagus is reached, in the mucous membrane of which they are rather far from one another. The blood vessels in the pharynx form a close network with elongated meshes, whence spring the capillaries which supply the rudimentary papillæ and the glands. The œsophagus is much less vascular. The nerves of the pharyngeal and œsophageal plexus contain ganglionic cells. The only terminations known are those in the muscular fibres.

II. Anatomical Lesions of the Pharynx and Œsophagus.

Pharyngitis.—Inflammation of the buccal mucous membrane and of the nasal fossæ generally shows a great tendency to spread to the pharyngeal mucous membrane; there are, however, numerous exceptions to this rule, for though inflammation of the tonsils and soft palate, such as variolic pustules, scarlatinal angina, &c., extends with the greatest facility to the posterior surface of the soft palate and the posterior wall of the pharynx, it is not the same with stomatitis, such as aphthous, lead, mercurial, ulceromembranous, and scorbutic stomatitis, which is generally limited to the lips, cheeks, and alveolar mucous membrane. Further, pharyngitis shows special anatomical peculiarities related to the structure of the pharyngeal mucous membrane. Among others we may cite, as a special lesion of the pharynx, granular pharyngitis.

Superficial or catarrhal inflammation of the pharynx is most frequently caused by a chill, and coincides with coryza or tonsillar

angina; it is characterised, as inflammation in every other mucous membrane, by redness and a muco-purulent secretion on the surface. In **variola**, the pustules developed in the pharynx are not as solid as those of the buccal mucous membrane; the epithelial layer which limits them is very easily detached, so that instead of well-formed pustules nothing is seen but white spots or patches composed of softened and desquamated epithelium mixed with a puriform mucus. In **measles** and **scarlatina**, points of redness are nearly constant in the pharynx; scarlatinal pharyngitis is particularly grave and acute, and it most often gives origin to a superficial exudation called a *croupous membrane.* The surface of the mucous membrane is covered by a pulpy, soft, white or greyish layer, which is composed of epithelial cells, thrown off in great numbers and mixed with muco-pus (pultaceous angina). In scarlatina and measles, moreover, a very serious form of pseudo-membranous angina is sometimes observed.

Erysipelatous pharyngitis resembles stomatitis (*vide* p. 183). Erysipelas is first developed in the skin, and extends to the pharynx either by the nasal fossæ or by the mouth, and if it descends to the lower part of the pharynx it may reach the epiglottis, the aryteno-epiglottic folds, and the larynx. It is rare for **mercurial poisoning** to cause irritation of the pharynx, unless it is by the local action of bichloride of mercury or the acid nitrate of mercury. We have, however, observed inflammatory œdema of the lower part of the pharynx and of the aryteno-epiglottic folds, coinciding with ulcerative stomatitis and glossitis in a case of subacute poisoning ending in death, caused by cauterisation of lupus of the face with acid nitrate of mercury. **Diphtheritic pharyngitis** often follows diphtheritis of the tonsils and soft palate; the pseudo-membranous plaques cover the posterior surface of the soft palate, and thence invade either the nasal fossæ or the posterior wall of the pharynx. When the diphtheritis passes, as often occurs, from the tonsils to the larynx, or from the larynx to the tonsils and nasal fossæ, the portion of the pharynx which separates these parts is often more or less affected by it, and the diphtheritic false membranes show the same structure as on the tonsils and soft palate (*vide* p. 205). **Typhoid fever** causes tumefaction of the closed follicles which are found at the lower part of the pharynx. These small tumours, which have the same appearance as the isolated and hypertrophied closed follicles of the small intestine in the same disease, are caused by infiltration of the follicles and neighbouring connective tissue with lymph cells.

The acuminated part of the small tumours first undergoes ulceration, and finally the whole of the new formation. These lesions generally coincide with the laryngitis of typhoid fever, already described (*vide* p. 46). The posterior wall of the pharynx is not as commonly affected by **syphilis** as the soft palate and the tonsils; sometimes, however, mucous plaques are found here, showing the same characters as in the buccal mucous membrane; they are accompanied with inflammation and granulation of the pharyngeal mucous membrane. Gummatous neoplasms are often developed beneath the mucous membrane at the level of the basilar process, and may acquire considerable size. The losses of substance, and the granulating ulcerations which they undergo during repair, are sometimes very extensive. As the result of these deep ulcerations, close adhesions or an actual union may take place between the posterior wall of the pharynx and the soft palate. The pharynx is then separated into two cavities; the upper situated above the soft palate and communicating with the nasal fossæ, the lower limited above by the soft palate and communicating with the mouth. If the soft palate is not perforated respiration can only take place by the mouth. Julius Paul has collected a great number of these cases ('Arch. de Méd.,' 1865, vol. ii.)

Granulations of the pharynx.—In all cases of acute pharyngitis, particularly those of diphtheria, scarlatina, &c., red granulations are seen projecting on the surface of the mucous membrane. They are blended with the mucous membrane, and are composed

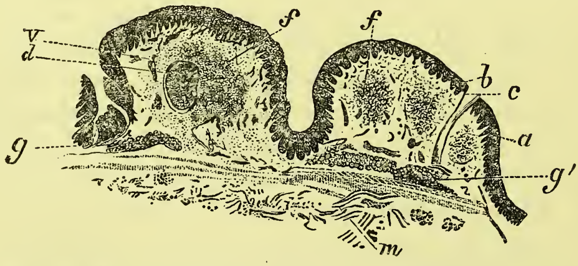


Fig. 100.—Granulations of the Pharynx, from a case of Diphtheritic Pharyngitis.

a, epithelial investment; *b*, papillæ; in the centre of each projection formed by the two granulations, closed follicles, *f*, *f*, are seen; *c*, excretory duct of an acinous gland seen longitudinally; *d*, transverse section of a glandular duct; *g*, *g'*, acini of mucous glands situated in the deep submucous layer; *v*, blood vessels; *t*, connective tissue; *m*, muscle fibres. Magnified 10 diameters.

of hypertrophied closed follicles, around which the mucous membrane is inflamed. On examining sections of these granulations

the thickened epithelium is seen, and the papillæ, beneath which the mucous corium is more or less infiltrated with lymph cells. The relief of the granulation is almost entirely caused by the closed follicle which occupies its centre; sometimes instead of a single follicle two or more contiguous ones are found. The excretory ducts of the acinous glands pass between the follicles or at their edges, sometimes even through the middle of one of them; but the glandular acini are more deeply seated in the submucous connective tissue below the part occupied by the closed follicles. In the granulations which accompany acute pharyngitis the follicles are crowded with lymph cells and are larger than normally, their tumefaction giving prominence to the granulation; but inflammation of the connective tissue of the mucous membrane also assists in the formation of a granulation.

Chronic pharyngitis.—Pharyngeal granulations are constantly met with in a rather frequent form of chronic pharyngitis, known under the name of granular or glandular pharyngitis (Guéneau de Mussy). This lesion, which is sometimes related to chronic cutaneous disease, and often to phthisis, or to the daily use of brandy, is most frequently due to a chronic process, characterised by the changes in the closed follicles just described and by alterations in the mucous glands of the pharynx. On the surface of the pharynx, which is generally congested, the acinous glands are seen to project more than normally. In the centre of the glandular swelling the excretory duct is often seen to be surrounded by a whitish lining, due to disaggregation and imbibition of the epithelial cells; the enlarged duct sometimes contains puriform mucus, and a superficial ulceration may be present at these spots. Similar inflammation is seen in the depression of the mucous membrane situated in the midst of the agglomerated closed follicles, found in the upper part of the pharynx. The closed follicles themselves are swollen. When the lesion is older, atrophy, following inflammation of the acinous glands and closed follicles, may be seen at certain points. The mucous membrane is irregular, thin in places, and shows small cicatrices, resulting from superficial ulcerations; it is thickened at other points from tumefaction of the acinous glands and closed follicles and of the surrounding connective tissue. The blood vessels, particularly the veins, are dilated and prominent, and true pharyngeal varices exist. The changes in the acinous glands may lead to cystic dilatations, formed at the expense of the acini and glandular ducts; small calcareous concretions may even be found. Chronic pharyngitis is some-

times related to the presence of tubercular granulations developed in the superficial connective tissue of the chorion. These granulations, the centre of which becomes rapidly opaque, may be the point of departure of ulcerations similar to the tubercular ulcers of the tongue. Isolated or confluent tubercles of the pharynx, and the more or less extensive ulcerations which result from them, are absolutely the same, from the histological point of view, as those already described in the soft palate (*vide* p. 198). The lesions of the acinous glands are identical. When tubercles are seated in isolated or confluent closed follicles of the pharynx, which is not rare, at the base of the tonsils and at the upper part of the pharynx, they resemble in development and character those of the tonsils (*vide* p. 211). The upper part of the pharynx, which is very rich in closed follicles, often shows inflammatory thickening of these follicles and of the mucous membrane itself. This tumefaction may be so considerable as to obstruct the Eustachian tubes and to cause either temporary or permanent deafness. This sometimes occurs after scarlatina and typhoid fever, and it is also observed in cretins. Certain cases of deaf-mutism seem to be caused essentially by obliteration of the Eustachian tubes from chronic hypertrophy of the lymph follicles (Klebs).

Retro-pharyngeal abscess may follow very acute inflammation of the mucous membrane; it has been observed in scarlatina. Sometimes it is caused by the local action of a caustic poison, or by foreign bodies deeply fixed in the pharyngeal mucous membrane setting up inflammation of the submucous connective tissue. More frequently, however, the pus of these abscesses is derived from the vertebræ and their periosteum (vertebral caries, cervical Pott's disease). The submucous abscess varies in size and shows a tendency to extend towards the lower parts of the pharynx, where it may cause œdema of the glottis, or along the œsophagus into the posterior mediastinum. It may open spontaneously into the pharynx, which is the most happy termination, but it may also open into the respiratory passages.

Œsophagitis.—It often happens that after death the cardia being relaxed, the substances contained in the stomach regurgitate into the œsophagus and cause cadaveric change. The epithelial investment is disaggregated and softened and forms a white, pulpy layer, and the muscular coat is sometimes of a greenish colour. **Superficial inflammation**, resulting from febrile exanthe-

mata, is less marked in the œsophagus than in the mouth and pharynx; such are the pustules of variola, which are seen at autopsies either as isolated or confluent and forming patches with sinuous edges, covered by a layer of dissociated epithelium and infiltrated with pus. In generalised pemphigus, plaques are sometimes seen in the œsophagus similar to those observed in confluent variola. The administration of an emetic may also determine small œsophageal pustules, followed by superficial ulceration. Inflammation of the œsophagus is sometimes met with in **scarlatina** simultaneously with pharyngitis. The pharyngeal eruption of measles may also extend into the œsophagus. Diphtheritis also sometimes invades the œsophageal mucous membrane; the diphtheritic false membrane must not, however, be mistaken for the equally white and pulpy patches composed of the mucidinæ of thrush mixed with epithelial cells. Another series of causes of œsophagitis is the ingestion of very hot fluids, or of irritant or corrosive fluids. When the caustic agents are brought into contact with the œsophageal mucous membrane, they, after imbibition of the epithelium and the connective tissue of the mucous membrane, produce scars which determine suppurative eliminative inflammation around themselves. After elimination the necrosed parts leave more or less deep and extensive ulcerations, the base of which generally extends as far as the muscular layer. The submucous connective tissue of the parts surrounding the ulcer and the connective tissue which separates the muscular fasciculi are infiltrated with embryonic cells. Later, if the disease undergoes cure, the cicatrix is formed by the organisation of fleshy granulations and by the formation of new epithelium on their surface. Gradually the newly-formed connective tissue shrinks, causing cicatricial contraction, and consequent narrowing of the œsophageal tube. Œsophagitis is sometimes caused by **foreign bodies**, which, by reason of their size, can neither be extracted nor pushed into the stomach. Ulcerations, abscesses, and even perforations may be the consequence. The œsophagus may also be perforated from without inwards, from the extension of an inflammatory focus having its origin in tubercular or caseous bronchial or tracheal glands. Abscesses of the mediastinum sometimes open into the œsophagus. **Syphilis** very rarely exerts its action on the œsophageal mucous membrane; many cases, however, exist with autopsies, in which cicatricial narrowing of the œsophagus has been attributed to syphilis. Fibrous contractions of the œsophagus are often mul-

tiple, and may be more or less extensive. At their level cicatricial bands are found, and a fibrous tissue which extends as far as the muscular layer; this is generally thickened at the same spots, which fact is due to an abundant new formation of connective tissue between the contractile fasciculi. The œsophagus is dilated above the obstacle, and this dilatation may take the form of a lateral diverticulum. The most common seat of contraction is at the level of the larynx and at the lower part of the œsophagus, above the cardia.

Tumours of the pharynx and œsophagus.—The **fibrous polypi** which project from the roof of the pharynx, and which spring from the bones at the base of the skull, have been considered with the nasal fossæ (vol. ii. p. 34). **Fibro-myomata**, small and round in form, and developed primarily in the muscular layer of the œsophagus, sometimes project from the mucous surface; they are composed of connective-tissue bundles and smooth muscle fibres; they are generally not larger than a pea and cause no accidents. Lipomata are also sometimes met with about the size of a nut, projecting in the form of polypi into the œsophageal cavity. In old subjects small warty, flat papillomata are frequently observed, covered with a layer of whitish pavement epithelium formed of smooth or composite papillæ. They vary in number and size, and greatly resemble warts on the back of the hand. **Mucous cysts**, developed from glands, have been recognised in the pharynx and œsophagus; they are hardly as large as a pea, though Klebs records having seen two in the œsophagus as large as a nut. Two cases of **dermoid cysts** of the pharynx and œsophagus have been recorded, and may be mentioned here on account of their rarity. **Tubercles**, which are seen rather frequently in the pharyngeal mucous membrane, are extremely rare in the œsophagus. **Carcinoma** of the pharynx is not common, and when it affects this organ it seems to commence either in the tonsils, or in the bones at the base of the skull, or in the glands of the neck. Whether scirrhous or encephaloid, carcinoma causes rapid infiltration of the mucous and submucous tissues, and forms a prominent tumour which ulcerates and gives origin to granulations formed of the morbid tissue. Its extension is rapid, and it soon invades the base of the tongue, the larynx, &c. The lymph glands of the neck are always affected. Carcinoma never seems to be developed primarily in the œsophagus, but it may reach this organ after originating in the pharynx, the cardia, the lymphatic ganglia, or

the connective tissue of the mediastinum. It causes organic narrowing, and more or less extensive fungoid ulcerations. **Pavement epithelioma** is seen in the pharynx as an extension of lingual epithelioma, but it is also developed rather frequently as a primary tumour. It commences in the mucous membrane itself. We once observed the participation of the mucous glands of the œsophagus in its development. These glands, which were tumefied at the edge of the new formation, and their saccules, enlarged and filled with pavement cells, were distinguished with difficulty from the neighbouring epithelial tracts belonging to the new growth. The epithelioma was composed, in this case and in many others we have observed, of large pavement epithelial tracts which had not undergone epidermic evolution (tubular pavement epithelioma). Lobulated epithelioma with cell nests is more frequent. Infiltration of the mucous tissue usually extends around the œsophagus in the form of a ring for a distance of from two to ten centimetres. The surface of the mucous membrane is white and anæmic, and the tumour on section is seen to be equally white and dry. The morbid mass, which is from five to eight millimetres or more in thickness, and which extends from the surface of the mucous membrane to the muscular layer, causes stenosis of the tube with rigidity. It is dilated above the contracted part. Later, when the tumour extends to the muscular layer and to the neighbouring parts, portions of the tube primarily affected ulcerate and food can pass down the œsophagus. The most frequent seat of cancroid is the middle third of the œsophagus, at the level of the bifurcation of the trachea. The neighbouring glands early undergo change and become infiltrated with epithelial elements. Sometimes they preserve their form and capsule and remain isolated, though they contain morbid tissue, which under the microscope is seen to be in every way similar to that of the primary tumour. These glands frequently attain a considerable size, even to that of a turkey's egg ; they are then entirely changed, softened at their centre, and sometimes, after having become solidly united to the trachea, they open into this tube. It often happens that the primary tumour compresses or alters the recurrent nerve and causes complete aphonia.

CHAPTER V.

THE STOMACH.

I. Normal Histology of the Stomach.

THE stomach has three coats; the mucous membrane, the muscular coat, and the peritoneum. They are united by connective tissue. This tissue is so loose beneath the mucous membrane that it enables this membrane to glide easily over the muscular layer. The mucous membrane is pink during digestion, and it is pale when the stomach is empty. It then falls into folds and rugæ, which is due to its slight elasticity and to contraction of the muscular coat. It is thicker immediately above the pylorus than at any other part. A section passing perpendicularly through the mucous membrane shows a layer of glands, grey in colour and thicker at the pylorus than at the great curve. The glands of the stomach are of two kinds: pyloric glands, found throughout the pyloric region and also met with, but in less numbers, near the cardia; and the peptic glands, which occupy the large curve and the middle part of the stomach. The former are said to secrete mucus, the latter gastric juice. On microscopically examining delicate sections of the stomach of a dog, cut perpendicularly to the surface, ramifying and parallel tubular glands are everywhere seen, opening into depressions on the surface. These small superficial depressions are separated by circular projections of the mucous membrane, which have the appearance of slightly raised and conical papillæ. These projections and depressions are everywhere lined with a single and uninterrupted layer of small cylindrical, caliciform cells, 20 μ in length, and which rest on the corium. In the lower part of these cells a nucleus is seen contained in a mass of protoplasm. The superficial, cup-like portion contains transparent mucus, which is continuous with the delicate layer of mucus generally spread over and adherent to the surface of the mucous membrane of the stomach. This superficial mucus has an acid reaction (Cl. Bernard). This superficial layer of cells and the

contiguous part of the submucous tissue alter rapidly after death under the influence of the gastric juice, which by continuation of its action digests these tissues. Thus the histological details of the gastric mucous membrane cannot be observed in man, except in individuals beheaded or hanged.

The peptic glands are formed of cylindrical tubes, which terminate in the depressions already mentioned and extend from these to the bottom of the glandular layer; they measure from 400 to 500 μ in length and 60 μ in width. They contain two kinds of cells, the peptic cells, described by Kölliker, which appear as dark granular spherical masses in the centre of which a small round nucleus is seen. These cells are located along the inside of the gland near its internal border, so that they project in the form of small swellings on the internal surface of the glands; they stain deeply with carmine and with soluble anilin blue. The other cells are small and polyhedric in shape, clear, very finely granular, and entirely fill the tube of the gland; these are the cells which are looked upon by Heidenhain as peptic cells, contrary to what has been hitherto admitted; but there is nothing to show that this new view is the more correct one. In sections cut parallel to the surface the large spherical cells are seen at the periphery of the glandular tube, while the small polyhedric cells advance towards the centre and almost block a very narrow lumen. Every tube is surrounded by a delicate layer of connective tissue, the fibres of which have the same direction as the gland, but the four or five tubular glands which open into the same depression on the mucous surface are isolated from similar neighbouring groups by a greater thickness of connective tissue. The **pyloric glands** of the pyloric region are also composite tubular glands, and resemble the peptic glands by their general arrangement, except that they are larger, the tubes wider, and that they contain only one variety of cells, namely, cylindrical cells. These cells resemble in structure those of the surface, but their free border is not generally cup-shaped, and they are only limited by a delicate cuticle; they are very long and narrow, and their nucleus is ovoid; the central lumen of these tubes is larger than that of the peptic glands. They compose the greater part of the superficial or glandular layer of the stomach. They are separated from one another by fasciculi of connective tissue, combined with smooth muscle fibres, towards the lower part of the saccules (muscular layer of the mucous membrane). On the surface of the corium, beneath the epithelium, there is an hyaline layer, or basement membrane,

beneath which numerous capillary vessels ramify and form a network continuous with that which surrounds the glands. This network is supplied by the arterioles which come from the submucous tissue, and they give origin to small veins which join larger veins situated in the same tissue. It is important to add that the arterioles which furnish the capillaries to the mucous membrane are derived from the coronaries, the splenic, the gastro-epiploica dextra, and the pyloric arteries; they all enter by the peritoneal surface and ramify in the successive layers of the stomach, so that the zone of distribution of each arteriole in the mucous layer forms a cone, the apex of which reaches the submucous tissue and the base the surface of the mucous membrane of the stomach. The lymphatics form, according to Teichmann, two networks, one situated beneath the glands, the other in the submucous tissue; the large trunks perforate the muscular coat at the level of the curves of the stomach. There is besides a superficial lymphatic network belonging to the peritoneum.

II. Pathological Anatomy of the Stomach. Lesions of Nutrition.

Anæmia.—Anæmia of the gastric mucous membrane seems to be unfavourable to the normal secretion of the gastric juice; thus this condition is, in all probability, generally anatomically coincident with dyspepsia in chlorosis.

Congestion and ecchymosis.—During physiological digestion the mucous membrane of the stomach is red and the superficial capillaries are filled with blood. Congestion of the gastric mucous membrane is seen pathologically at the commencement of and during the course of acute catarrh, as well as in all forms of inflammation of the stomach. On opening a congested stomach at an autopsy made twenty-four hours after death in winter, the surface of the stomach will be seen beneath a layer of cloudy mucus to be red, or marbled bright or dark red, or of a slate colour. The mucous membrane is often marked by small bright red spots, very near together; at other times the islets of congestion are larger, the colour of which varies from the bright red of arterial blood to the dark red of venous blood. The congestion is always much more marked at the summit of the rugæ than in the grooves which separate them. It seems as if the contraction of the muscular tunic, which produced these folds in the last moments of life, had strangulated their bases and retained the blood contained

in the vessels at their apices. Small round lenticular patches of congestion are also found at a higher plane than the anæmic tissue of the surrounding mucous membrane; sometimes a slight depression is seen at their centre without there being any loss of substance, this depression being simply caused by contraction of the subjacent smooth muscle fibres. Side by side with these congested and red patches others are found of a slate-grey colour or spotted with black.

In sections cut perpendicularly to the surface of a mucous membrane previously hardened, at the level of the congested parts, the superficial capillaries and those surrounding the glands as well as the small veins of the mucous membrane are found to be distended and filled with red blood corpuscles. Sometimes even there has been extravasation of the red blood corpuscles and infiltration of these elements in the connective tissue, in which case true ecchymoses are produced. The brown or slate-coloured staining of the congested patches and the ecchymoses of the mucous membrane are due to the transformation of hæmoglobin into hæmatin. This change occurs much more rapidly in the stomach than in any other organ, for it is affected after death by the action of the gastric juice on the red blood corpuscles contained in the vessels or those which have been extravasated. This change may be even seen to take place when examining a congested stomach for two or three hours, patches which were originally red turning brown or slate-coloured. The pigmentation of the mucous membrane, either in the form of spots or of tree-like branches corresponding to the veins, may be produced during life. These alterations consecutive to repeated congestion are very common in chronic gastritis where it is accompanied with profound changes of the connective tissue and the glands. The brown or black granules which result from destruction of the red blood corpuscles penetrate the cells with which they are in contact; thus the connective-tissue cells of the mucous membrane, and even the epithelial cells contained in the glands, are themselves deeply pigmented in the slate-coloured patches. If the circulation has been arrested in consequence of repletion of the vessels and extravasation of the blood in a superficial ecchymosis, the gastric juice may effect here an actual digestion of the mucous surface which has been deprived of its means of nutrition. This is one of the causes of ulceration, which we will consider further on when considering simple ulcer of the stomach.

Cadaveric softening of the gastric mucous membrane.—It is im-

portant not to refer to a pathological cause the cadaveric changes which the stomach undergoes after death. If when life ceases this organ contains, together with food, a marked quantity of gastric juice, this acts on the mucous membrane, deprived of life, and partly digests it; the phenomena are, in fact, the same as in artificial digestion in a vessel. This digestion is also aided by the central position of the stomach, which is placed between the spleen and the liver, at the point where the temperature is high and is kept up for a certain time after death; this digestion is hence naturally more rapid in hot weather. When thus softened, the mucous membrane is found to be in a condition of pulpy detritus if a current of water be brought to play on it. For a long time this condition, actually post-mortem, was described as inflammatory, and the names of white, red, or slate-coloured softening were given to it. This peculiar condition of digestion of the mucous membrane is met with in the most dependent parts of the stomach; it is very marked in children who have died while the stomach was full of milk, which by rapidly undergoing lactic fermentation constitutes a favourable condition for cadaveric digestion of the stomach. Hunter long ago indicated this action of the gastric juice after death, which did not, however, prevent pathologists at the beginning of this century from regarding this softening as due to inflammation, particularly the condition called gelatiniform softening by Cruveilhier. It is possible that congestion and inflammation play some part in this softening, but it is mainly due to cadaveric decomposition.

Lesions of the glands.—In chronic gastritis both atrophy and hypertrophy of the glands are produced, and will be described when treating of this disease; but we may indicate here a lesion of the glands which occurs in **phosphorus poisoning**, and which cannot be considered to be inflammatory in character, since it is solely characterised by fatty degeneration of their epithelial cells. It is well known that under the influence of this poisoning the epithelial cells of the lung, liver, and kidneys, the muscular fasciculi of the heart and muscle fibres, both striated and smooth, undergo rapid and marked fatty degeneration. The same degeneration may affect the epithelial cells of the gastric glands. They are seen to contain a large number of fatty granules; the gland itself seems also to be entirely filled with them. Senftleben, who first observed this condition of the gastric mucous membrane, and being indoctrinated by Virchow with the view that primary fatty degeneration is of an inflammatory nature, referred this change

to gastritis caused by the direct action of the phosphorus. It is indisputable that in certain conditions, which cannot always be determined, phosphorus causes primary inflammatory lesions of the gastric mucous membrane; it is probable that in these cases it forms phosphoric acid and that this caustic agent acts directly; but, as we proved long ago, phosphorus itself does not irritate the tissues into which it is introduced. Thus if a fragment of phosphorus be introduced beneath the skin, or into a serous cavity, it causes no inflammation around itself. We do not wish, however, to deny that primary fatty degeneration of the glands of the stomach may not become the point of departure of ulceration; it is easily understood, in fact, that those parts of the mucous membrane which have undergone degeneration and mortification may be attacked by the gastric juice and become the starting point of an ulcer. But fatty degeneration of the glands of the stomach is not only observed in phosphorus poisoning; it is sometimes met with in alcoholic subjects, and also in those in whom it is difficult to determine the cause. It is seen in the form of whitish opaque spots, which affect a certain resemblance to patches of thrush; on examining these under the microscope in sections cut perpendicularly to the surface, fatty degeneration of the glands and of the epithelium is seen. Beside these spots ulcerations or small scars may be observed; the latter result from the direct action of the gastric juice on the mucous membrane, which is no longer protected by its epithelial lining. On examining these scars under the microscope the blood vessels are found to be filled with a brown substance, which is nothing else than coagulated blood changed by the action of the gastric juice.

Lesions of the vessels.—It is not unusual to meet with a more or less large artery of the stomach in an atheromatous condition coincident with a similar lesion in the aorta. It is conceivable how this alteration may, by provoking vascular obstruction, become the cause of ulceration of the mucous membrane. We have also frequently observed amyloid degeneration of the arteries which ramify in the walls of the stomach; this lesion coincides with a similar degeneration of the arteries of the intestine.

Inflammation of the Gastric Mucous Membrane.

Acute catarrhal gastritis.—It is almost impossible to appreciate in man the slight degrees of gastric catarrh which, in all pro-

bability, constitute the anatomical lesion in certain forms of dyspepsia. The epithelial cells which line the surface of the mucous membrane fall soon after death, owing to their being attacked by the gastric juice, which has no action on them during life; generally no trace of them can be found twenty-four hours after death. The changes observed in the glandular cells are, for the same reason, of doubtful value. The superficial connective tissue of the mucous membrane is equally altered. Thus to comprehend these lesions we have to depend upon experiments and the study of lesions produced in the stomachs of animals. If irritation of the gastric mucous membrane has been produced in a dog by injection of an emetic into the veins, this membrane will be found to be deeply injected at places and covered with a mucous or mucopurulent secretion. It may be examined at once or after having been treated by a hardening and fixing fluid, such as alcohol, Müller's fluid, or osmic acid. The gastric mucus is milky and fluid, and it contains a large number of leucocytes, flakes formed of epithelial cells, isolated cells, and red blood corpuscles. Sections of the congested parts, cut perpendicularly to the surface from specimens hardened in alcohol and microscopically examined, show very marked distension of the capillaries of the superficial network which occupies the upper part of the interglandular projections. These projections, which in section appear as small villosities, are more prominent than normal, and are swollen and club-shaped. This is due to distension of their capillaries. Around the vessels, moreover, in the subepithelial and œdematous connective tissue, extravasated red blood corpuscles and leucocytes are seen. When the inflammation is subacute the cells of the epithelial investment remain attached at certain points; at others, on the contrary, the epithelium is entirely detached: this occurs at the prominent extremities of the interglandular folds, which are highly congested. The depressions of the mucous membrane into which the glands open are narrowed or even completely effaced by tumefaction of the connective tissue which surrounds them; these depressions are nevertheless still lined by their epithelial cells. Leucocytes are not found in the narrowed necks of the glands. As to the glands themselves, neither the pyloric nor peptic glands show any change; not only do they contain no leucocytes, but both their peptic and cylindrical cells are normal. From what precedes it will be seen that artificial gastric catarrh consists essentially in congestion of the surface of the stomach, in repletion of the superficial vascular network, in extravasation

of the serum containing red blood corpuscles and lymph cells, and in œdematous and ecchymotic tumefaction of the interglandular projections ; but the glands of the stomach do not seem to play any part in it, or at all events a very fugitive *rôle.*

In man, it is generally quite impossible to recognise the histological lesions of a similar condition ; but redness of the mucous membrane and the ecchymoses observed very distinctly in the best marked cases, added to the characters of the gastric fluid, indicate superficial catarrh of the stomach pretty correctly. We have frequently observed the histological characters of catarrhal inflammation of the human stomach when preserved under quite exceptional circumstances, as in cases of pulmonary phthisis, variola, and typhoid fever. In hardened sections the superficial epithelium was found to be preserved at places and all the cells to be caliciform. At the base of these cells there was a layer of round cells. The superficial capillaries were turgid and filled with blood. On the surface of the mucous membrane the adherent layer of mucus contained filaments and balls of mucus, and a few lymph cells. In a number of infectious febrile diseases, such as puerperal fever, variola, &c., pallor and opacity of the glandular layer of the stomach are observed, which is more or less mammillated. The cells of the glands are filled with fine proteic or fatty granules, and on section are seen under a low power to be opaque. All the cells touch each other, and are rather indistinct when examined in the same gland ; isolated they are seen to have the same globular form as the peptic glands. The granules generally become pale on the addition of acetic acid ; others, which disappear under the action of alcohol or ether, are obviously of a fatty character. In the most prominent part of the papillæ the glands are hypertrophied. This is a condition of tumefaction accompanied with granulo-fatty degeneration of the epithelial cells of the gland, similar to that seen in the liver and kidneys of the same subjects. It has been called glandular gastritis by the Germans (Virchow, Klebs). It is probable that cadaveric decomposition plays a certain part in this alteration of the gland cells, but it is impossible for us to clear up this question, as the law does not permit of autopsies being made till twenty-four hours after death. In the post-mortem examination of a case of typhoid fever, in which the stomach had been very well preserved, the connective tissue situated immediately beneath the glandular saccules was found infiltrated with a considerable quantity of lymph cells. This lesion was recognised by the naked eye from the difficulty with which the mucous membrane

glided over the muscular coat. In a section cut perpendicularly to the surface of the stomach a whitish and opaque line was seen between the glandular layer and the muscular coat. Sections examined under the microscope showed a considerable collection of round cells in the whole of the cellular tissue situated beneath the saccules. The intermediate tissue between the glands and muscular coat was also much thickened. This infiltration of lymph cells extended between the glands on the one side and the muscular fasciculi on the other. Throughout the gastric mucous membrane the capillaries and veins were extremely dilated. Dilatation of the vessels of the stomach, and particularly infiltra-

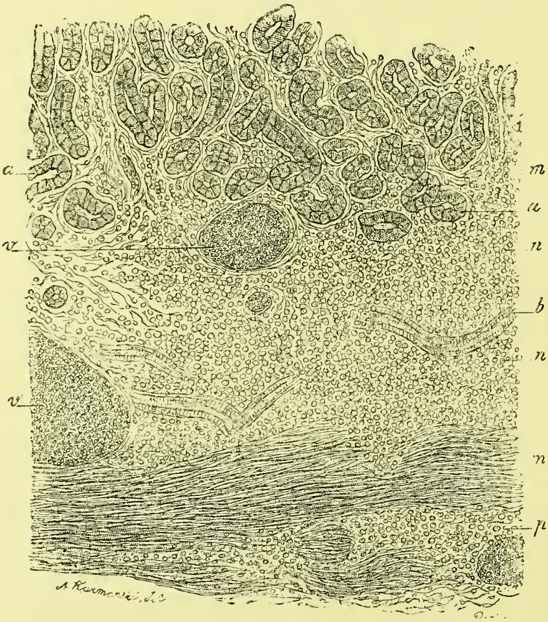


Fig. 101.—Connective Tissue of the Mucous Membrane of the Stomach, from a case of Typhoid Fever.

a, a, gastric glands cut across; *v, v,* capillaries and small veins, dilated and filled with red blood corpuscles; *b,* small arteries; *n, n,* lymph cells filling the meshes of the cellular tissue situated beneath the glandular saccules and constituting a large band interposed between the latter and the muscular layer, *m.* Lymph cells are also seen to be extravasated between the glandular saccules and the muscular fasciculi.

tion of this organ by lymph cells in typhoid fever, are lesions comparable to those of Peyer's patches in the same disease. M. Chauffard has observed many cases.

Chronic gastric catarrh.—The lesions of chronic gastritis are more extensive than in acute gastritis, and are not limited to the

superficial layer of the mucous membrane, but may invade the glands and the mucous corium; the muscular coat is also frequently changed. In cirrhosis of the liver in drunkards, and in all diseases which accompany obstruction of the vena porta, as in some cardiac conditions, the mucous membrane is often found to be thickened, red, brown, or slate-coloured. This thickening, which is slight in some places, and forms at others circumscribed greyish elevations separated from one another by superficial grooves, gives the mucous membrane a mammillated appearance. If at these points sections be cut passing through a papule and the groove limiting it, the glands at the upper projecting part of this papule will be seen to be much dilated and filled with a more or less granular epithelium. The attenuated part at the level of the groove is, on the contrary, remarkable for its atrophied glands, which are smaller and narrower than normally, and have thickened walls, which contain free fat granules or a few granular cells. The papules are yellow and opaque when the glands contain many fat granules, which granules are made very apparent if the mucous membrane be hardened in osmic acid. The submucous connective tissue is everywhere thickened, particularly so at the level of the grooved lines which circumscribe the papules. The colour of the mucous membrane is grey, red, or slatish at places, according as the mammillated condition, the congestion, or the consequent pigmentation predominate. Pigmentation may affect the epithelial cells of the glands; in fact, in certain cases of alcoholic gastritis, partly atrophied glands may be seen at the deeply pigmented spots containing small epithelial cells filled with black pigment.

In almost all cases of chronic and mammillated gastritis prominent brilliant spots may, on careful examination, be observed on the surface; they are mucous and transparent, and resemble small air bubbles; these are cysts formed of the spherically distended glands filled with a viscous mucus. This substance is so adherent to the walls of the gland that it does not escape spontaneously, even though the orifice be free, though narrowed and of a button-hole shape. On pressing one of these cysts between two glass slides, mucus exudes from the orifice. The walls of these dilated glands are lined with caliciform epithelium, and the small cysts, the diameter of which may reach one millimetre, or even more, contain a few vesicular spherical cells in the midst of the mucus. These cysts are generally surrounded with glands which show, either in their necks or saccules, one or more dilatations small

enough to escape macroscopic observation. The mucous membrane is at the same time covered with a thick layer of grey viscous and very adherent mucus. If chronic inflammation continues for some time, the surface of the mucous membrane granulates,

FIG. 102.—CYSTIC DILATATION OF THE GLANDS OF THE STOMACH.
(Figure borrowed from Virchow.)

and the fibro-vascular tissue, which separates the glands, pushes out fleshy granulations toward the free surface in the form of villi containing vascular loops. The cylindrical epithelium cannot be seen on the surface of these papillary growths, for it always falls twenty-four hours after death. Round or irregularly polygonal cells are only found; they are generally granular. When these granulations are about half a millimetre in height, they look like villi and the gastric mucous membrane resembles that of the small intestine. In patients who have died during the process of digestion these growths are often found to be filled with fat granules. They are generally found near the pylorus, but they

may be seen over the whole or a great part of the surface of the mucous membrane. If chronic inflammation persists, the villi increase in size and may even become united at their bases, their extremities remaining free on the surface of the hypertrophied

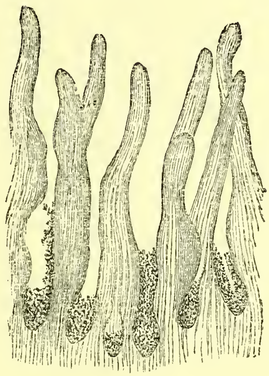


Fig. 103.—Newly-formed Papillæ developed on the Gastric Mucous Membrane and taking the villous form. Magnified 60 diameters.

mucous membrane. One consequence of this new state of the papillæ is obstruction of the excretory ducts of the glands. These are atrophied either entirely or partly, and by epithe-

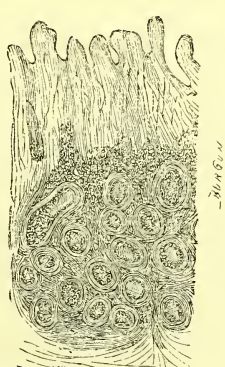


Fig 104.—Cylindrical-celled Adenoma of the Stomach, with Villous Condition of the Surface of the Tumour. Magnified 20 diameters.

lium continuing to reform in their interior they take a more or less spherical form, become isolated and dilated, and often when examined in sections have the appearance of small cysts. The

internal walls of the cysts are lined with caliciform cells, and their lumen is filled by a fluid containing spherical cells. In other cases

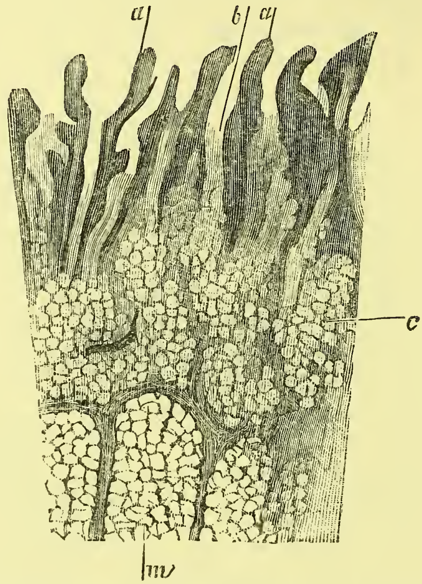


FIG. 105.—SECTION OF A GLANDULO-PAPILLARY POLYPUS OF THE STOMACH.

a, newly-formed papillæ; *b*, glandular ducts; *c*, saccules; *m*, deep saccules. Magnified 40 diameters.

these glands send expansions into the deep surface of the mucous

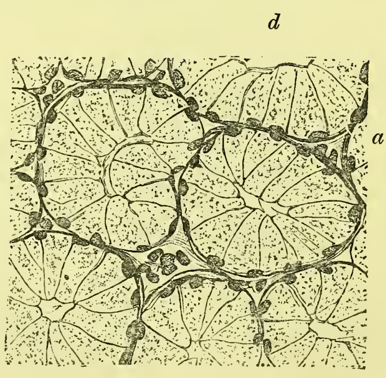


FIG. 106.—SACCULES REPRESENTED AT *m* IN FIG. 105. Magnified 300 diameters.

a, *c*, septa of the saccules with nuclei of the epithelial cells; *d*, mucous cells.

membrane, where becoming isolated they may develop into true cysts

similar to those just described. The thickness of the mucous membrane is thus markedly increased at certain points. From this partial thickening of the mucous membrane a small sessile tumour may result, which eventually becomes pediculated if able by its prominence and weight to pull upon the surrounding mucous membrane. A polypus is thus produced which varies in nature and consistence according as it is composed of fibrous tissue and papillæ (fibrous and papillary polypus, villous polypus of Cruveilhier), or of mucous cysts (mucous polypus), or of these various kinds united. The mucous polypi are remarkable for their transparency and softness.

Chronic gastritis, characterised by glandular dilatations and small polypi, is much more frequent in old persons than in young subjects. We have, however, seen a case in which a large number of mucous cysts were present all over the gastric surface. This was in a young woman who died from gastro-enteritis following typhoid fever. The seat of mucous polypi near the pylorus explains the accidents to which they may give rise, as, for instance, obstruction of the pylorus; sometimes they may even project into the duodenum. The centre of the largest of these poylpi is generally composed of smooth muscle fibres ; these will be described when treating of tumours of the stomach.

Diphtheritic gastritis.—This is very rare, very limited, and only occurs in generalised diphtheria.

Pemphigus of the gastric mucous membrane.—In many cases of general pemphigus, blackish, superficial ulcerations are found on the gastric mucous membrane ; they seem to be produced by the same cause as bullæ on the skin. In colour they are often very dark, for they are the seat of small hæmorrhages which become black when acted upon by the gastric juice. The blood still present in the vessels is generally coagulated, broken down, and of a black colour. We have often observed these ulcers in specimens brought to the Anatomical Society of Paris, and in one case they were covered with a brown detritus containing a large number of spores and tubes of the oïdium albicans. These ulcers soon undergo cicatrisation, and in the cicatrix Chalvet has found deposits of subnitrate of bismuth accumulated during the active period of ulceration.

Phlegmonous gastritis.—This very rare lesion is generally looked upon as a secondary or metastatic inflammation occurring in typhus, septicæmia, purulent infection, puerperal fever, and in

generalised purulent peritonitis. It may be caused by the local action of irritant or caustic fluids. The interglandular and submucous connective tissue of the stomach is infiltrated by a thick phlegmonous pus, which floods the lacunæ, but which is rarely collected into foci; this purulent infiltration invades the connective tissue interposed between the muscular layers and subserous tissue, and is thus propagated by the peritoneum, which frequently becomes inflamed throughout its whole extent. If a section of the surface of the stomach be examined, the different layers will be seen to be separated by a layer of pus. The thickened gastric walls are rigid and do not collapse. In a case we published, the stomach was infiltrated with pus throughout its whole extent, and its rigid walls were at certain places a centimetre in thickness. There were two superficial ulcers of the mucous membrane and general peritonitis. In this form of gastritis the mucous membrane may finally become thin and perforated, when small openings are seen, through which the pus escapes.

Lesions produced by corrosive and irritating agents.—The introduction into the stomach of corrosive sublimate, sulphuric acid, ammonia, potash, &c., is followed by the formation of brown or black scars, around which the deeply injected mucous membrane soon shows all the phenomena of eliminative inflammation. The fall of the scab leaves a loss of substance, a perforation followed by peritonitis. Arsenious acid, according to most writers, is also said to produce ulcerative gastritis. But it is probable that the initial lesion consists in infiltration of the mucous surface, the glandular layer, and connective tissue with blood, and that gangrene and elimination of the eschar follow the ecchymoses. In a recent case of poisoning we observed so marked an infiltration of blood that the red and mammillated mucous membrane was in places nearly a centimetre thick. In sections the red colour was seen to be due to the presence of blood; red blood corpuscles were everywhere inside the glands, the epithelial cells of which were, however, preserved; they were also in the interglandular connective tissue, and between the fibres of connective tissue beneath the glandular saccules. The subglandular tissue was particularly full of blood and markedly thickened by the extravasation. The capillaries and small veins of this tissue were greatly dilated and filled with red blood corpuscles. In the parts of the mucous membrane thus altered, circulation of the blood and nutrition of the elements became impossible; gangrene thus occurred in the diseased part and

gradually spread. It is probable that all the cases of gangrenous gastritis published are due to poison. Klebs, however, reports the case of a child who died after gangrenous inflammation of the pharynx with tumefaction of the cervical glands, and on opening the stomach many round gangrenous spots were found surrounded by a zone of purulent eliminative inflammation. It should be remarked that in malignant anthrax of the tongue in the pig similar lesions are observed in the stomach.

Simple ulcer of the stomach.—Simple ulcer of the stomach was first described by Cruveilhier, who distinguished it from cancerous tumours. Simple ulcer is characterised by a loss of substance which is more or less regularly circular in shape; the edges are sharply defined, the mucous membrane being arrested at the limit of the ulcer without showing a border with a granulating or reversed surface. The base of the ulcer is pale, fibrous, and grey, and, as it is subjected to the action of the gastric juice, it generally has a superficial layer undergoing molecular destruction. This loss of substance is, in fact, due to actual digestion of a limited part of the stomach in which circulation of the blood has completely ceased. Simple ulcer commences by superficial erosion of the mucous membrane only. The mucous corium is then invaded and destroyed, and the base of the ulcer is finally formed of eroded muscular fasciculi; these fibres themselves gradually disappear till of the wall of the stomach only the peritoneum remains; when this is finally destroyed the gastric cavity is bounded by the neighbouring organs with which the peritoneum has contracted adhesions. Thus the liver, pancreas, and lymph glands, more or less eroded by the gastric juice, may form the base of a gastric ulcer. After the mucous membrane, the mucous corium and the muscular layers have been eliminated by progressive molecular destruction, a large patch of ulceration is produced, the edges of which are sloping, the base being smaller than the opening; the ulcer is then funnel-shaped, with the large end turned towards the free surface of the mucous membrane. In explanation of this form, which is moreover not constant, it is supposed that the ulcer corresponds to a limited region supplied by an arteriole. An artery which springs from the peritoneal surface of the stomach, and divides and subdivides in the muscular layers, the submucous coat, and the mucous membrane, has the form of a cone with its base turned towards the mucous membrane. If the part destroyed by ulceration is that supplied

by a small artery, it results that in a deep ulcer which affects all the coats of the stomach the mucous membrane will be more extensively ulcerated than the submucous and deeper tissues; hence the funnel-shaped form of the ulcer. Sometimes, however, the ulcer is flat with irregular edges. At the edges and base of the ulcer one or more moderate-sized arteries are generally found, which have been entirely cut across by the ulcerative process; their lumen is sometimes obstructed by a plug of newly-formed connective tissue. Patients affected with this lesion often die from severe vomiting of blood. The cause of this accident is easily discovered, as the ulcerated artery which yielded the blood of the final hæmatemesis is found to contain a post-mortem coagulum. The size of simple ulcers of the stomach varies greatly; Cruveilhier describes one which measured 160 millimetres at its largest diameter, and which extended from the pylorus to the cardia along the lesser curve. In this case the base of the ulcer was formed by the liver, pancreas, and arch of the colon. Where the lesion is seated along the great curve or along the anterior surface of the stomach it may cause perforation and general peritonitis, which may be localised by means of adhesions. The ulcer is sometimes single, sometimes multiple. It may be seated either on the lesser curve, on the pylorus, the cardia, the posterior surface, and more rarely on the large curve, at the great cul-de-sac, or on the anterior surface of the stomach; it may even invade the lower part of the œsophagus, and it sometimes developes primarily in the duodenum.

On making a section of the wall of an ulcer it is seen to be formed of the pre-existing tissue; the connective tissue is here only slightly thickened, but there is no juice, as in cancer, nor anything that recalls the neoplasm of a tumour. If the glandular layer be examined at the edge of an ulcer, where it is well preserved, the glands are seen to be much elongated, which is due to the fact that the connective tissue of this layer is thicker and richer in cells than normally. The epithelial cells of the gland are perfectly preserved. Beneath the glands the connective tissue is thickened, and here at the edge of the ulcer the vessels are constantly found to have undergone sclerous hypertrophy of their walls and narrowing of their calibre. These vascular lesions differ according to the nature and diameter of the vessels and according to the part examined; thus in the section of an arteriole of a certain size obliterating endo-arteritis will be seen at the point where it is divided and plugged on the surface of the ulcer, and in

the neighbouring parts endo-arteritis will be found with fleshy granulations projecting into the lumen of the vessel. Further on the vessel is filled with coagulated blood. Much smaller arteries show an irregular and marked thickening of their walls. It is the same with the capillaries. In one case we found a few lymph vessels of the submucous connective tissue filled with lymph cells; here also the connective tissue showed colloid change in places, and at these points white, black, and reticulated fibrils of connective tissue were seen limiting very narrow areoli containing a colloid substance and a few rather large round cells. When the ulcer has invaded and partly destroyed the muscular coat, fasciculi of smooth fibres forming irregularly cut tufts of filaments, composed of dissociated contractile elements, will be seen on its surface under the microscope. The tissue bordering the loss of substance seems to be formed of normal elements, or to be infiltrated with fine fat granules and undergoing degeneration. The fibres of the muscular tissue beneath an ulcer sometimes also contain fine fat granules, and in the arterioles the fibrous septa, which separate the contractile fasciculi, as well as those in the peritoneal connective tissue, are altered in the same way as in the submucous tissue. If hepatic tissue forms the base of the ulcer, interstitial hepatitis is seen at this spot. In a rather large number of cases the peritoneum becomes perforated and the stomach communicates with an intraperitoneal abscess which is limited by adhesions and situated on the posterior surface of the stomach, or between the stomach, liver, spleen, and diaphragm. Barth records a case of simple ulcer on the anterior wall of the stomach, in which the loss of substance was replaced by the anterior wall of the abdomen and by the posterior surface of the xiphoid appendix, which, deprived of its periosteum, was eroded and even destroyed at certain points. In fact, all organs which come in contact with the gastric juice are attacked by it; the pancreas seems to be able to resist better than the liver. Cruveilhier states having observed simple ulcers opening into the transverse colon, into the third part of the duodenum, and, still more extraordinary, an ulcer communicating across the diaphragm with the left bronchus.

The anatomical diagnosis of simple ulcer of the stomach is not difficult if the foregoing details be remembered, namely, the absence of a raised border, the state of dryness and hardness of the base of the ulcer, and the absence of juice on section of the tissue which forms the base and edges, differentiate it from carcinoma and all other tumours.

The cure of gastric ulcer is possible. Rather frequently small cicatrised ulcers are found, either isolated or near to partly or fully developed ulcers. The cicatrix of small superficial ulcers which have undergone cure is bordered by mucous membrane puckered by contraction of the fibrous tissue; but the part primarily ulcerated is not covered by mucous membrane, and shows neither glands nor epithelium. If, however, a layer of epithelial cells had been present on its surface during life, they would not be found twenty-four hours after death. Larger cicatrices may become the seat of fresh ulceration (Cruveilhier), so that ulcers, which no longer reveal their presence by any symptom such as pain or hæmatemesis, and which might be looked upon as cured, may, in consequence of some indiscretion in diet, or simply by suppression of the milk diet, become the seat of most alarming hæmatemesis and of perforation. **The fatal termination of this disease is due** either to excessive hæmorrhage from one of the large vessels at the base or edge of the ulcer or to perforation of the stomach. These serious accidents, particularly perforation, are incomparably more frequent in simple ulcer than in carcinoma of the stomach. The arteries which may be affected by ulceration and occasion hæmatemesis are, in the first place, the splenic artery, then the branches and trunk of the pyloric and coronaries, and finally the gastro-epiploicæ.

What is the cause of simple ulcer? As destructive ulceration takes the form of the territory supplied by an artery, it is natural to refer the molecular mortification of this tissue to embolism or thrombosis of one of its vessels. This is the hypothesis put forward by Virchow, who supports it by a certain number of clinical cases and by the experiments of L. Meyer. Very marked atheroma of the gastric arteries has also been observed by ourselves and by Fœrster; this seems to be itself a sufficient cause of ulceration by the hindrance to the circulation which results; but it is not the usual cause, and particularly in the case of young subjects. The quality of the food, changes in the gastric juice, substances which have a local action on the stomach, such as alcohol, mercury, &c., may also be considered in the etiology of this disease. An ulcer once produced, it is supposed that the continuous action of the gastric juice, joined to sclerosis of the small arteries, diminishes the blood supply and the nutrition of the diseased part; complete cicatrisation is thus prevented and the series of accidents observed ensues. A case of ulcer of the stomach has been recorded in trichinosis (Ebstein).

Simple or perforating ulcer of the duodenum.—Simple ulcer of the duodenum greatly resembles that of the stomach by its anatomical characters, by the state of its edges and base, and by its tendency to progressively destroy the walls of the intestine, which form its base, and the organs with which it is in contact. It is much more frequent in men than in women, in the proportion of ten to one (Krause); it is generally seated in the first part of the duodenum and simultaneously on the pylorus; it is more common in the anterior than in the posterior half; it is often associated with hindrance to the flow of the bile and pancreatic juice. When it terminates by recovery and cicatrisation it causes, even when not seated near the pylorus, contraction and stenosis of the pyloric ring, with all its consequences (dilatation and hypertrophy of the stomach, vomiting, &c.) Ulcers of the duodenum cause hæmorrhage and perforation. The hæmorrhage is due to erosion of the arteries contained in the ulcer, namely, the pancreatico-duodenal, the gastro-duodenal, the gastro-epiploica, the hepatic arteries, and the vena porta. Perforation occurs most frequently on the peritoneal side, and thus causes localised adhesive peritonitis, which unites the duodenum to the neighbouring organs; but foci of limited suppuration, sacs, and fistulous tracts communicating with the base of the ulcer may also be present. Thus an ulcer, the wall and base of which were formed by the liver and pancreas, communicated with the gall bladder; purulent and fistulous tracts have also been observed communicating with the ulcer, following the course of the ribs, and opening by an abscess under the skin into the scapular region or along the vertebral column (Krause). Perforation may occur between the stomach and the second or third part of the duodenum in consequence of simple ulcer of the stomach.

III. Tumours.

Lipoma.—This form of tumour is rare and not remarkable; it may project either under the serous or under the mucous membrane.

Sarcoma.—Primary sarcoma of the stomach is rare. Virchow relates a case in which it was seated on the lesser curve and affected all the coats; at the same time, however, sarcoma of the ovaries and peritoneum existed.

Papillary tumours and adenomata have already been described when treating of hypertrophy of the glands in chronic gastritis.

Lymphadenoma.—This is sometimes found in leukæmia and

adenia. These growths have the same appearance as carcinomata and form soft white granulating tumours and yield a milky juice; they ulcerate at their centre, and microscopic examination alone indicates their true nature. They may occupy a more or less considerable extent of the mucous membrane and may attain one to two centimetres in thickness. On dividing them perpendicularly to their surface the different layers of the mucous membrane may be recognised. Before ulceration occurs the glandular layer is preserved, the glands of which seem longer than normally; this is owing to the increased thickness of the connective tissue which surrounds them. This tissue is infiltrated with lymph cells which are arranged in longitudinal series between the fibrous fasciculi surrounding the glands, the glandular epithelium of which is preserved. Beneath the glands the submucous connective tissue is also extremely thickened, and is equally infiltrated with lymph cells, which on being brushed out of delicate sections the adenoid reticular tissue can be distinctly seen. Lymph cells are also seen between the muscular fasciculi; they are here fewer in number than in the connective tissue, and are interposed between the layers of longitudinal and transverse fibres. When the glandular layer is destroyed by ulceration irregular fleshy granulations take its place.

Tubercle.—Tubercle is very rare and is only met with in general tuberclar ulcerations of the whole intestine. It has the same appearance and pursues the same course in the gastric as in the intestinal mucous membrane.

Calcareous grains.—Under this name Virchow has described a change which consists in infiltration by calcareous salts of a limited portion of the mucous membrane and of the glands seated in its substance; ulceration may follow this infiltration and cause superficial destruction of the mucous membrane.

Fibro-myoma.—Tumours composed of fasciculi of unstriped muscular tissue and connective tissue are sometimes met with in the stomach. They are comparable as regards their structure and development to myomata of the uterus. Springing from one of the muscular layers of the stomach, they may project either towards the mucous or the serous surface. 1st: A myoma which projects under the mucous surface is generally seated near the pylorus; if it becomes large it may be drawn by the food through this orifice and may project into the duodenum. We showed a case of this kind at the Société Anatomique; it was a hard fibrous polypus, about the size of the thumb, 8 c.m. in length, covered by the gastric mucous membrane and projecting into the middle of the

first part of the duodenum; the mucous membrane was ulcerated at the point where it passes through the pylorus, owing principally to constant friction. The gastric mucous membrane, which covered the pylorus, was sprinkled over with mucous cysts, visible to the naked eye. Finally, as we have already shown, these polypi, which are partly muscular, partly fibrous, and partly composed of hypertrophied glands, may be mucous in appearance. 2nd: The polypi composed of muscular fasciculi and fibrous tissue, which project into the peritoneal cavity, are generally hard and small; they may, however, attain the size of an almond or nut, and sometimes they become calcified.

Syphilitic tumours and ulcers of the stomach.—Pathologists and syphiliographists (Virchow, Leudet, Lancereaux, &c.) have recorded many cases of hypertrophic thickening of the walls of the stomach, and of ulcers due to syphilis; but most of these cases are rather doubtful. The same may be said of the case observed by Klebs, in which he simultaneously found gummata of the liver and ulcers of the stomach and intestine. We have ourselves observed a case of syphilitic tumours of the stomach coinciding with very characteristic gummata of the liver. Along the small curve and near the pylorus the gastric mucous membrane was raised by flattened, moveable tumours, one of which was 5 c.m., the other 3 c.m., and the third 2 c.m. in diameter; at these spots the mucous membrane was preserved, but was thin and adherent. In a section cut perpendicularly to the surface, the indurated submucous tissue was found to be from 8 to 12 mm. thick; it was perfectly distinct from the subjacent muscular layer. The figure 107, A, drawn the natural size, represents a section passing through the different layers of one of these tumours of the stomach. The curve *b* of the tumour was depressed, though covered by the mucous membrane *a*. The muscular layers *g* and *h* were normal or hardly thickened, while the submucous connective tissue *f* was greatly hypertrophied. This connective tissue, fibrous in consistency and very dense, yielded no juice on scraping; in colour it was yellow. The muscular layer was hypertrophied. The pylorus was narrowed. The stomach had not contracted adhesions with the liver; but at the lesser curve it was adherent to indurated lymphatic glands, and the peritoneal surface showed a hard, white, radiating cicatrix. Under the microscope the glandular layer seemed to be covered with small villous or papillary projections, perpendicular or oblique to the surface. These villi were formed by the hypertrophic granulations of connective tissue surrounding the

tubular glands (B, *a*, fig. 107). These glands were separated from one another by thickened fibrous connective tissue; their ducts were narrowed, specially at the centre of the mammillated growths (B, *b*, fig. 107). The terminal saccules of the glands were almost normal in the greater part of these small tumours; they were seen in sections at a millimetre or a millimetre and a half beneath the surface, in the form of circles (B, *c*, fig. 107), having almost

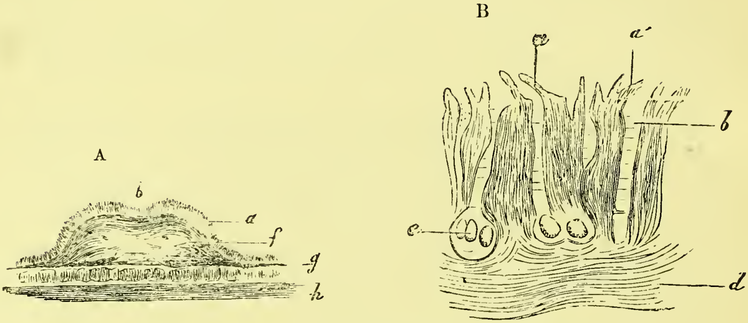


Fig. 107.—A, Section of a Gumma, drawn natural size. B, Section of a Glandular Layer of the Stomach at the Level of a Syphilitic Gumma.

a, *a*, interglandular projecting villi; *b*, glands; *e*, glandular saccules; *d*, connective tissue of the mucous membrane. Magnified 20 diameters.

the diameter of normal saccules; at the centre of the tumour they were, however, rarer and smaller. Under a higher power (from 200 to 400 diameters) the villi seemed to be formed of a close connective tissue, covered with a few flat cells, and containing between their fibres round embryonic cells and fat granules. The tubular glands contained small cubical epithelial cells, forming an incomplete lining. The terminal dilatations of the glands were partially filled with mucous or caliciform cells. The corium, which constituted, as has been said, all the relief made by the new formation, was hard, dense, felt-like, and was well supplied with arterial and venous capillaries filled with blood; it contained elastic fibres and connective tissue, between which many small round or slightly elongated embryonic cells were present. Round islets of true embryonic tissue were found moreover in the midst of a granular and slightly abundant ground substance. At the depressed centre of the tumours the deep connective tissue was directly continuous to the surface; for at this point the atrophied glandular layer was reduced to a few widely separated tubes and groups of saccules. The muscular layers were thickened and transparent; the unstriped fasciculi

were separated by bands of fibrous tissue intermixed with small round embryonic cells. But these elements were not found in the interior of the fasciculi between the muscle cells, which latter were large, transparent, refractile, and contained their characteristic nuclei. The same new formation of cellular elements was present in the layer of peritoneal connective tissue. This case, in which the lesions were characterised by flattened tumours seated in the submucous connective tissue, and showing the structure of fibrous gummata, left no doubt as to the syphilitic nature of the neoplasm. After its consideration it is difficult not to admit the existence of syphilitic ulcers of the stomach. In the same case the lymph glands situated above the lesser curve were large, white superficially, and infiltrated with juice (*vide* vol. i. p. 558).

Carcinoma of the stomach.—Cancerous tumours of the stomach, considered from the clinical point of view, that is to say, tumours characterised by gravity, extent of ulceration, propagation to the lymph glands, and by secondary growths, are very common, and show the same naked-eye appearances and pursue the same course, although tumours quite different in structure may be distinguished in this group. Encephaloid carcinoma of the stomach, for example, cannot be distinguished with the naked eye from cylindrical-celled carcinoma, which is very common here. The varieties of carcinoma observed in the stomach are in order of frequency: encephaloid carcinoma, fibrous or scirrhous carcinoma, colloid and melanic carcinoma. They are almost always seated near the pylorus or lesser curve; sometimes also they are found at the cardia, from which they extend to the larger curve and fundus. They show a great tendency to extend over the posterior wall, and they sometimes surround almost the entire stomach. Carcinomatous tumours commence simultaneously in the submucous tissue and in the glandular layer. They then form protuberances which increase progressively in breadth and thickness. If a section of one of these tumours be made perpendicularly to the surface, the glandular layer is seen to be thickened and slightly transparent; but the principal mass which causes the relief of the new formation is composed of submucous connective tissue. Here, as in much smaller tumours, such as may be observed in the secondary growths of carcinoma developed primarily in the breast, a milky juice is obtained on scraping the divided surface. Under the microscope the submucous connective tissue already shows alveoli filled with newly formed cells, while the glandular layer shows elongated

glands filled with small cylindrical or cubical cells. The elongation of the glands is due to budding of the connective tissue which separates them. This tissue contains a large number of round embryonic cells between its fibres, and papillary processes are sent out, which may even project beyond the orifices of the glands. This process, in chronic gastritis as well as in tumours of the stomach, is common to all new growths in the gastric mucous membrane. Near the cancer the mucous membrane of the stomach is generally of a red or violet colour; it is sometimes softened or mammillated, and shows the signs of chronic inflammation, with marked pigmentation. Small mucous cysts may also be observed, produced by distension of the glands. When the tumour is not ulcerated it is generally of a white colour, and it is prominent, mammillated, or spread over the surface of the mucous membrane. The subsequent ulceration varies much in extent; it may occupy the whole of the lesser curve. At the pylorus it often takes the form of a ring; when this occurs the course of the food, which had at first been obstructed by the tumour, becomes temporarily re-established. The edges of the ulcer are raised, projecting, and sometimes eroded for a certain extent; its base is generally fungoid, bleeding, filled with detritus, or, if the cancerous mass be deeply seated, the muscular coat seems to be partly denuded, or even perforation may be produced; but this is incomparably rarer in carcinoma than in simple ulcer. The muscular coat is always hypertrophied round about a tumour; this hypertrophy may in fact extend and affect the whole stomach, as occurs when the new growth is seated at the pylorus. The stomach often contracts adhesions to the neighbouring organs. On its peritoneal surface miliary carcinomatous nodules, or round plaques of the same character, are often seen, which have set up localised adhesive peritonitis. The adhesions thus established with the liver, pancreas, and diaphragm are often thick and partly composed of cancerous tissue. They limit the ulceration and prevent the gastric juices escaping into the peritoneal sac. Sometimes the stomach is quite empty, but most frequently a thick black fluid is found in it, resembling wet soot or coffee dregs; it is similar to what is vomited in these cases. The dark colour is due to blood modified by the gastric juice. The lymph glands of the lesser curve are always partly or entirely changed. Finally, secondary nodules of cancer are often found in the neighbouring organs, in the liver in particular, where they are seen as small cancerous patches in the midst of the hepatic tissue, and show on examination the same

structure as the primary tumour of the stomach; sometimes, however, they form a mass in the liver at the spot where the stomach ulcerates.

Varieties of carcinoma of the stomach.—These are, in order of frequency, encephaloid, scirrhous, colloid, and melanic carcinoma.

Encephaloid carcinoma, almost always primary, appears generally in the form of an ulcer, with a granulating and irregular surface and raised everted edges, which are composed, as well as the base of the ulcer, of a soft vascular tissue, whitish or pink in colour and rich in milky juice; sometimes, however, part or the whole of the mucous membrane is carcinomatous. On microscopical examination an alveolar stroma is found, as in every other tumour of this nature, with large polyhedric or globular cells, of various forms, which constitute the milky juice. The secondary nodules in the liver rapidly attain a considerable size in this form of carcinoma; the entire liver may be invaded and become a great size. These large tumours in the liver contrast with the cancerous ulcer in the stomach, which is relatively small, and which might lead one to think at first that the cancer of the liver was primary; but it is not so, as may be clinically demonstrated.

Telangiectasic or hematode carcinoma, which is only a variety of encephaloid carcinoma, is seen in the stomach in the form of soft and highly vascular fleshy granulations, in which some of the vessels are often considerably dilated in an irregular manner (*vide* fig. 108). The facility with which the veins are altered and attacked by the neoplasm is remarkable. On examining the stomach on its peritoneal surface, at the point where the mucous membrane is ulcerated, large dilated veins may be noticed passing towards the liver. These veins are sometimes filled with a white, extremely soft and pulpy tissue, resembling that of which the primary tumour is composed. It is vascular, and the vessels are derived from the walls of the veins. This blocking of the veins by vascular cancerous granulations, the vessels of which are also irregularly dilated, may extend as far as the portal vein and its hepatic branches. **Primary scirrhus of the stomach** is more rare than encephaloid. The tumour, which is hard, poor in juice and blood vessels, has a thicker fibrous stroma than that of encephaloid carcinoma, but it resembles it in its seat, its mode of development, and its propagation in the form of nodules to the peritoneum, the glands, the liver, and the pancreas. The development of the morbid tissue is followed more easily in

scirrhus of the stomach than in encephaloid, for the tumour is better able to resist cadaveric changes. It often forms small

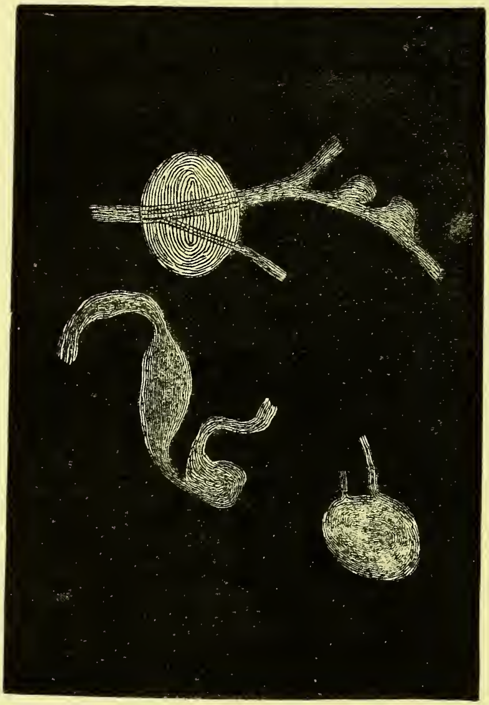


Fig. 108.—Aneurismal Dilatation of Capillary Vessels in Encephaloid Carcinoma of the Stomach. Magnified 50 diameters.

secondary nodules in the stomach, covered by the mucous membrane. In sections of these nodules, cut perpendicularly to the surface, it is seen that the carcinomatous alveoli are first formed in the submucous connective tissue, beneath the glandular saccules, which are more or less hypertrophied.

Colloid carcinoma is rather frequent in the stomach; it is characterised by the gelatiniform appearance of the tissue which composes it. Care must, however, be taken not to include among carcinomatous tumours all which are colloid in appearance, for cylindrical-celled epithelioma often undergoes more or less extensive colloid degeneration.

Epithelioma.—Cylindrical-celled epithelioma is the most common of the primary tumours of the stomach, formerly called

cancerous. It has the same naked-eye appearances as encephaloid carcinoma: the same form of ulcer with raised everted edges; the

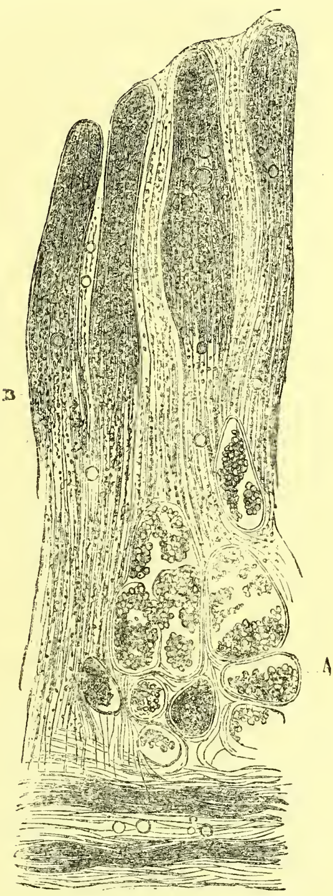


Fig. 108.—Villous Hypertrophy of the Connective Tissue of the Gastric Mucous Membrane, as may be observed in all Tumours of the Stomach, or at their borders (Adenoma, Papilloma, Carcinoma, Epithelioma).

A, glandular saccules; B, connective tissue separating the tubular glands. Magnified 80 diameters.

same vascular granulating surface covered with a thick opaque white fluid; the same tissue rich in milky juice forming the base and edges of the ulcer; the same gravity, and likewise secondary nodules in the liver, and secondary infection of the lymph glands of the lesser curve of the stomach. It is seated by preference at the pylorus, in the region of the mucous glands. On microscopical

examination, however, nothing is easier than to determine its exact nature. In hardened sections deep depressions are seen, having the general shape of the mucous glands, and which are lined with cylindrical epithelium; these tubes are, however, more irregular than mucous glands, longer, wider, and are often seen in the form of cavities, from the surface of which papillæ or blood vessels bud out, covered by the same epithelium (vol. i. p. 273). These growths take their origin in the mucous glands on the surface, and progressively invade the deeper layers. At the edges of the ulcer greatly elongated mucous glands are seen, and a new formation of small round cells in the connective tissue interposed between the glands (fig. 109). The lymph glands of the small curve were always degenerated in the cases we have observed, and in sections of these glands cavities were found the shape of which seemed to be determined by the lymph channels; these cavities were everywhere lined with cylindrical epithelium, and villosities were seen on their surface covered by the same epithelium. The secondary nodules of the liver and other organs were also composed of a tissue containing tubes or small cylindrical or spherical cavities, and were always lined with the same form of epithelium. In the stomach the primary tumour often undergoes colloid degeneration either partially or throughout its whole mass, so that at first sight it might easily be mistaken for colloid carcinoma. In the parts which have a gelatinous appearance, cells are found which are either round, or intermediate between the spherical or cylindrical shape. They are filled with a transparent substance and the débris of elements; the cavities which they line have, at the same time, lost their cylindrical form to become spherical. The true nature of the growth is recognised in those parts of the tumour which have not undergone change, and in the glands and secondary nodules. If it is wished to study the development of cylindrical-celled epithelioma of the stomach, a recently-formed nodule of the tumour must be selected, and one which has not undergone considerable post-mortem change. In hardened sections it will then be seen that certain gastric glands have been pushed out in various directions into the deeper tissues; some, having passed through the muscular coat of the mucous membrane, have reached the submucous tissue, and have there formed numerous epithelial buds and alveolar cavities lined with cylindrical cells. **Lobulated pavement epithelioma** is very rarely met with in the gastric mucous membrane. It is always secondary either to epithelioma of the œsophagus which

has extended to the cardia or to epithelioma of the mouth, tongue, or œsophagus. Klebs has observed three cases of it: in the first there were secondary nodules in the cardia, consecutive to pavement epithelioma of the œsophagus seated at the level of the division of the trachea; in the second case a tumour of the large curve of the stomach was secondary to epithelioma of the face, the antrum Highmori, and the pharynx; the omental glands were equally affected; and in the third case five or six nodules were seated in the posterior part of the stomach, and were secondary to epithelioma of the back of the tongue. In these three cases the secondary growths in the gastric mucous membrane had the same characters as the primary tumours.

Hypertrophy of the muscular coat.—This change, generally caused by chronic gastritis, is described here because it almost always accompanies all tumours of the stomach, and is, moreover, very often mistaken for a tumour, particularly when located at the pylorus. In section, the enlarged muscular coat is seen as a pale grey mass, rather hard, semitransparent, fleshy, through which pass thick parallel layers of connective tissue, which give it a *cloisonné* appearance. This hypertrophy may be limited to the area of the cancerous mass, or may affect the whole of the muscular coat of the stomach. In some cases collected in the 'Recueil de la Société Anatomique' death was caused by considerable hypertrophy of the muscles of the stomach, accompanied with stenosis of the pylorus. This lesion often seems to be primary when neither tumour nor ulcer can be discovered in the stomach after death; but it is most probably always consecutive either to gastric catarrh or to a small and cicatrised ulcer. When such a cicatrix is seated near the pylorus, which is frequently the case, this orifice becomes narrowed, and in consequence of this narrowing the stomach undergoes enormous dilatation. On examining the smooth muscle fibres of the hypertrophied layer, they are found to be thicker and longer than in the normal condition. Hypertrophy of the muscular coat of the stomach may be caused by exaggerated action of the muscles, from the irritation produced by the presence of foreign bodies; this was the cause in two cases published by Luschka, who had found the shells of *gryphea cymbrium* in the stomach. When the hypertrophy is seated near tumours, trabeculæ of connective tissue, varying in thickness, are found between the muscular fasciculi; they contain tumefied connective-tissue cells and a

more or less considerable number of lymph cells. The contractile fibres are also hypertrophied. Here we have œdematous and inflammatory tumefaction of the muscular coats caused by the presence of the tumour: but hypertrophy is not always caused by the effort of the muscles to overcome an obstacle, for it may be present when there is no stenosis of the pylorus.

CHAPTER VI.

THE INTESTINE.

I. Normal Histology of the Intestine.

The small intestine.—The small intestine is composed of many layers of membranes, which are from without inwards: the peritoneum, a muscular layer formed of two layers of smooth muscle fibres, the first having a longitudinal direction, the second circular; and finally the mucous membrane and the cellular tissue which fixes it to the muscular coat.

The mucous membrane of the small intestine, continuous with that of the stomach at the pylorus, forms in the duodenum and jejunum semilunar transverse folds, or *valvuli conniventes*; the entire mucous surface is covered by villi, which give it a velvety appearance, easily seen with the naked eye on examination under water. These villi, extremely numerous in the duodenum and jejunum, decrease gradually towards the ileum. Throughout its whole extent the mucous membrane contains simple tubular glands, or the glands of Lieberkühn. In the first part of the duodenum racemose glands, or Brunner's glands, are also found, and throughout its whole extent isolated or agminated closed lymph-follicles. The corium of the mucous membrane is covered superficially with villi, and it is hollowed by numerous tubes in which the glands are lodged. Beneath the glandular saccules the muscular layer is found proper to the mucous membrane; it is composed of two planes of smooth muscle fibres, one longitudinal and the other transverse in direction; it is quite distinct from the outer muscular coat, from which it is separated by loose connective tissue.

The epithelium which lines all the free surface of the mucous membrane is formed of cylindrical cells, adherent one to another; they have oval nuclei, and their free edge is covered by a rather thick and striated plate; between them chalice cells are found. Letzerich, who discovered these cells without knowing that they were present in a great number of epithelial investments, looked

upon them as funnel-shaped organs intended to absorb emulsified fat in the intestine; but it has been demonstrated by numerous observers, among whom we may cite Eimer and F. E. Schulze, that these cells secrete mucus in their interior and are veritable unicellular glands. They are of the same height as the cylindrical cells surrounding them; they are swollen, globular, and contain a clear, transparent mucus, which escapes by an orifice above. This orifice, looked at direct, appears to be regularly circular, but seen in profile it seems to be edged by a defined border; often a drop of mucus continuous with that which fills the cellular cavity may be seen to exude; the nucleus of the cell is pushed down to the lower part of the cell, where it is embedded in a mass of protoplasm.

The intestinal villi, which are lined by the preceding cells, are thin, and measure from 0·2 millimetre to 1 millimetre in length. Near the surface they have a capillary network supplied by one or two arterioles which give off small veins. In the midst of the connective tissue of the villi, longitudinal unstriped muscle fibres are seen, by means of which shortening of the villus can be brought about. At the centre, there are one or two lymph vessels which terminate in culs-de-sac, and which are called lacteals, for they are the most important agents in the absorption of the chyle. The fat granules which are carried away by the lacteals first pass through the cylindrical epithelial cells and then through the connective tissue of the villus. The villi, by multiplying the absorbing surface of the intestine, also play a very essential part in this absorption.

Brunner's glands, situated in the first part of the duodenum, form a close layer from the pylorus as far as the orifice of the ductus choledochus. They are less frequent in the rest of the duodenum. They are racemose glands, are visible to the naked eye, and resemble histologically the salivary glands. **The tubular glands**, or **glands of Lieberkühn**, are present over the whole surface of the small intestine, and are only absent at the spots where the lymph-follicles project on the surface of the mucous membrane; here the glands and villi are both arrested, so that the closed follicle seems to be lodged in a depression of the mucous membrane. The glands of Lieberkühn are regularly cylindrical and tubular, and each one terminates in a cul-de-sac situated in the depth of the mucous membrane; they open between the villi. They have a mean length of 300 μ. They do not seem to have any proper membrane, but only a layer of flat cells which serve to

divide them from the surrounding connective tissue (Henle). They are lined by a layer of cylindrical cells which differ from those of the villi in that they have no striated border. Numerous chalice cells are found near the neck of the gland, but they progressively diminish in number and disappear altogether in the saccule. These glands assist in the secretion of the mucus which lubricates the intestine, and they elaborate the intestinal fluid.

The **closed follicles** of the intestine are lymphoid organs, analogous to those at the base of the tongue and to the tonsils; they are composed of a reticulate connective tissue imprisoning lymph cells. These follicles are sometimes isolated, as occurs in the jejunum, ileum, and colon, sometimes grouped together in patches situated opposite to the attachment of the mesentery, in which case they are called **Peyer's patches.** These patches, which are easily recognised, for here the mucous membrane is thickened and looks opaque on being examined by transmitted light, are generally elliptical in shape; their long axis, which corresponds to that of the intestine, is many centimetres in length. They are found in the ileum, but are most developed at the lower part of the small intestine. Their extent varies in different subjects. The isolated follicles are round; but crowded together in a Peyer's patch their largest diameter is perpendicular to the surface of the mucous membrane. They are composed of an adenoid tissue very rich in blood capillaries, and are separated from the connective tissue of the mucous membrane by a lymph sinus, across which the blood vessels pass.

The fatty matters and digestive juices contained in the small intestine are absorbed by the intestinal villi, and pass into the lymphatic system of the mucous membrane, which is composed not only of the lacteals of the villi and the sinuses of the closed follicles, but also of a very extensive network of lymphatics which surround the glands of Lieberkühn and communicate with the circumfollicular sinuses. It is from this system that the moniliform lymph vessels take origin, which pierce the muscular wall of the intestine and communicate with the lacteals of the mesentery.

The blood vessels form close networks in the villi, around the tubular glands, and in the closed follicles. The muscular layers have capillary networks proper to themselves, the meshes of which correspond to the direction of the fibres, longitudinal in the longitudinal layer, and transverse in the circular layer.

The nerves, which are derived from the sympathetic system, form

two plexuses in the intestine. The first, discovered by Meissner, is seated in the submucous connective tissue; it is composed of ganglia and pale fibres which are distributed to the smooth muscle fibres of the villi and of the mucous membrane, and may also form part of the nerve supply of the glands. The second, discovered by Auerbach, is the myenteric plexus, and is situated between the two layers of the muscular coat of the intestine. It is also formed of non-medullated fibres anastomosing and forming plexuses at the nodal points of which ganglionic cells are found. The meshes of this plexus are much narrower than those of Meissner's plexus, and the trabeculæ and ganglia are flatter.

The large intestine is constructed on the same scheme as the small intestine, but in man the mucous membrane has neither villi nor Peyer's patches. The closed follicles are larger and less numerous than in the small intestine, with the exception of the ileo-cæcal appendage, which is crowded with them. The glands of Lieberkühn are present all over the surface of the large intestine, with the exception of the spots occupied by the closed follicles. They have the same structure as those of the small intestine, only slightly larger. Beneath the tubular glands are the two layers of muscle fibres proper to the mucous membrane. The lymphatics are less numerous than in the small intestine; the blood vessels and the two plexuses of nerves have the same general arrangement.

II. Pathological Histology of the Intestine.

Cadaveric lesions.—As in the stomach the small intestine shows important cadaveric changes 24 hours after death. In the majority of cases it is pale, the mucous membrane colourless and covered with a thick, opaque deposit, which is easily detached. This deposit is formed chiefly of desquamated epithelial cells mixed with the mucus which normally exists on the surface of the intestine at the moment of death. Its opacity is given by epithelial cells, which fact may be ascertained by the microscope. The glands of Lieberkühn have also altered, and their cells have disappeared from the necks of the glands, so that they appear shorter than they are in reality. The superficial connective tissue of the mucous membrane is often softened; it may even be digested, so that the intestine breaks down from the least strain, as may be observed in summer weather, and particularly in children who have succumbed to diarrhœa. Sometimes even perforations,

which are entirely post-mortem, may be produced. One may rest assured that there has been no textural lesion during life when the thinned and perforated mucous membrane shows neither redness nor inflammatory infiltration. The vessels, when filled with blood at the moment of death, are often seen to be of a brownish or grey colour, owing to changes of the contained blood, due to the action of the intestinal juice.

Congestion of the intestinal mucous membrane.—Congestion of the intestinal mucous membrane is observed in most diseases of the intestine and in those caused by blood stasis in the vena porta. It is characterised by repletion of the blood vessels, which persists after death, and by a more or less abundant secretion of modified intestinal juice. On examining the congested parts under the microscope, the capillaries of the villi are seen to be filled with blood, which does not occur in the normal condition, and the superficial capillaries forming networks around the tubular glands are equally surcharged. Twenty-four hours after death the intestinal mucous membrane is often of a brown or grey colour. This staining is not always post-mortem; in fact, at the spots where this discolouration is marked a large quantity of brown or black pigment granules are often found in the connective tissue of the villi, which have themselves lost their epithelium. This lesion is constant in acute intestinal catarrh which has lasted a certain time.

Catarrhal inflammation of the intestinal mucous membrane.—Catarrhal inflammation of the intestinal mucous membrane is very common. It causes an increased secretion of juice; very different causes produce it, and the quality as well as the quantity of the fluid secreted varies in different cases. We will study the *modus operandi* of some of these causes. Purgatives generally act by producing irritation of the mucous membrane. It is true that the action of some of them, saline purgatives, for example, must be allowed to be purely physical, and that the secretion of a large amount of fluid in the intestine is due to osmotic phenomena. It has been sought also to explain the action of purgatives (Thiry and Radziejewsky) by irritation of the muscle fibres of the intestine, the peristaltic movements of the intestine and the rapid propulsion of the fluids preventing their absorption by the mucous membrane. Moreau was able to produce an abundant secretion by cutting the nerves which were distributed to an

intestinal loop. His method of procedure was as follows: Having drawn a loop of intestine outside the abdomen through an abdominal incision, three ligatures were applied so as to divide the loop into two equal segments. All the nerves leading to one of these segments were then cut in the mesentery. The whole was then replaced in the peritoneal cavity, and at the end of a few hours it was ascertained that the segment with the nerves cut contained much fluid, while the other did not contain any. The fluid had neither the characters nor the physiological properties of intestinal juice. It contained a large number of lymph cells and red blood corpuscles, which showed that the secretion had been produced by active irritation. Moreau also produced abundant intestinal secretion in an intestinal loop comprised between two ligatures, after having injected sulphate of magnesia. In this case the secretion seems to have been produced by endosmotic action. The action of drastic purgatives cannot, however, be explained in this way; they probably act by causing catarrh of the intestine. More acute and purulent catarrh can easily be produced in animals by the injection of irritant substances into the rectum. In young cats, into which we injected nitrate of silver or tincture of iodine, the mucous membrane was found 24 or 26 hours afterwards to be covered with muco-pus. The cylindrical epithelium was almost everywhere *in situ*, both on the surface of the mucous membrane and in the glands; the larger part, however, and in some places the whole of the cylindrical cells, both on the surface and in the glands, were converted into chalice cells. No endogenous formation of cells could be found, nor division of the nuclei, from which it is most probable that all the round cells in the purulent fluid had escaped from the blood vessels. The lymph cells were, in fact, numerous in the superficial connective tissue of the mucous membrane.

In man, the bad quality of the food taken, the excessive eating of fruit, or the want of appropriateness in the food to the age of the individual—for example, the want of human milk for children at the breast—the influence of cold, errors in diet, and indigestion, are the usual causes of simple catarrhal diarrhœa. Lower organisms (bacteridia), developed during lactic or butyric fermentation, are often found in large numbers in the fæces, but these bacteria are constantly present in the stools in the physiological condition. In these different cases the diarrhœal liquid passed by the patient is watery, stained yellow or brown by the bile, and contains fæcal matter. It generally contains very

few cylindrical cells of the intestine. In suppurative peritonitis, and particularly in puerperal peritonitis, the small intestine, which is bathed in the fibrino-purulent peritoneal exudation, is of a milky-white colour, and the mucous membrane is colourless and opaque; it seems to be infiltrated with pus, and is covered by a thin puriform layer. This is purulent catarrh caused by vicinity. In one form of chronic catarrh of the intestine, and particularly of the colon, a certain quantity of semiliquid, transparent mucus is produced in contact with hard fæces, which act as irritant bodies and become surrounded with a layer of mucus. At other times the intestinal secretion has all the microscopic characters of pus, and the voided fæces are covered by a layer of puriform mucus. This mucus, more or less tenacious, is passed in the form of false membranes or long filaments, and may be mistaken at first sight for fragments of the mucous membrane or for parasites. This transparent or opaque mucus always contains a quantity of cylindrical cells from the intestine and a variable number of lymph cells. In these forms of chronic catarrh the amount of the secreted fluid is in relation to the irritation of the mucous membrane. It is rare for simple catarrh of the mucous membrane to be accompanied with ulcerations, even superficially situated.

In chronic catarrh of the intestine a series of changes are observed in the tubular glands, which sometimes atrophy in places, sometimes hypertrophy, and take on, as in the stomach, the appearances of mucous cysts. The glands are hypertrophied in those parts of the mucous membrane where the irritation of the connective tissue being intense, the latter has developed in the form of villous processes between the glands. In the colon, for example, where villi do not normally exist, interglandular connective-tissue growths may be seen projecting from the surface of the mucous membrane in the chronic catarrh of children. Here, as in the stomach, the glands increase in length as the connective tissue surrounding them developes, and soon processes covered by epithelium resembling villi may be seen between the glands; the thickened portion of the mucous membrane forms a relief, and later may become pediculated by dragging on the neighbouring and normal mucous membrane. At other times hypertrophy of the glands is the predominating feature, and ends in the formation of mucous polypi, or in irregular slackening of the glandular layer of the colon, as we once saw in a phthisical subject. It is thus that the papillo-glandular polypi are produced in the rectum

and other parts of the intestine which are so frequent in children, and which sometimes become the starting-point of an invagination. The mucous contents of the dilated and cystic glands give the name of mucous polypi to these small tumours. They are quite similar to mucous polypi of the stomach, the structure of which has been described above (p. 249). The cells contained in hypertrophied tubular glands are always chalice-shaped and are filled with mucus.

Catarrhal inflammation of the intestine has received different names, according to the seat of the lesion; such as duodenitis, ileitis or inflammation of the ileum, typhlitis or inflammation of the cæcum, colitis or inflammation of the colon, and proctitis or inflammation of the rectum.

Simple or catarrhal **duodenitis** is rarely seen alone. Tumefaction of the second part of the duodenum, and particularly of the spot where the bile duct enters, results in retention of the bile and icterus.

In **ileitis**, or inflammation of the lower part of the ileum, tumefaction of the isolated closed follicles or of Peyer's patches is constantly seen, as well as the usual anatomical lesions of intestinal catarrh. This tumefaction has received the name of **psorenteria**. This lesion is chiefly observed in cholera, typhoid fever, in most of the infectious diseases, in the exanthemata, puerperal fever, &c. The isolated follicles, which normally cause no sensible relief, become developed and prominent like small round seeds; they are two or three times larger than normally, their surface is pale or

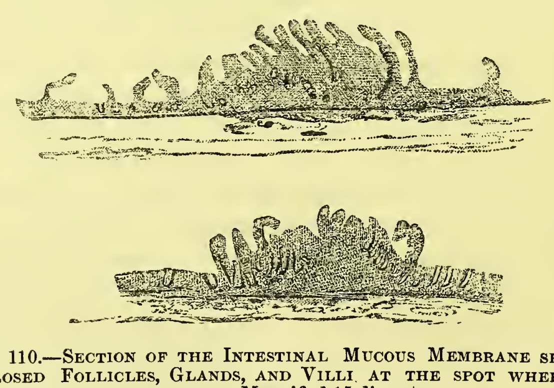


Fig. 110.—Section of the Intestinal Mucous Membrane showing Closed Follicles, Glands, and Villi at the spot where the follicles are inflamed. Magnified 15 diameters.

pink in colour, and on section they show a grey or pink semi-transparent tissue. On removing one of these small tumours

with the scissors, and on examining it under a low power, its prominent surface will be found to be partially covered by villi and mucous glands, which it pushes before it in developing. On examining delicate sections of these parts, cut after hardening the intestine, it will be seen that the closed follicles are hypertrophied, and that this hypertrophy is due to distension of the meshes of the retiform tissue composing them with lymph cells; in preparations obtained by means of scraping, flat, swollen, granular endothelial cells, containing two or more nuclei, will often be seen together with the lymph cells. This tumefaction of the isolated closed follicles, which is found in most forms of diarrhœa, is seen more distinctly at the end of the ileum than in other parts of the intestine, for the closed follicles are here generally more numerous; but it may be observed throughout the whole extent of the intestinal tract, in the colon as well as in the small intestine. When psorenteria is very marked, as occurs, for example, in cholera, hypertrophy of the follicles may terminate in ulceration, as has been observed by Bouillaud and Cruveilhier. Ulceration occurs, as in typhoid fever, when the special lesion falls primarily on the closed or agminated follicles; that is to say, a portion of the follicle projecting into the intestine undergoes mortification.

Typhlitis and perityphlitis.—Typhlitis is the inflammation of the vermiform appendage of the cæcum. When very acute it is generally accompanied with peritonitis, so that localised peritonitis or perityphlitis may be described together with typhlitis, of which it is the consequence. No part of the intestinal tract is so exposed to arrest or stagnation of fæcal matter or foreign bodies as the vermiform appendix; in fact, shaped as it is as a cul-de-sac, it is difficult for fæcal matter which has once found its way in to be expelled; contraction of the smooth muscle fibres of the appendix is the only force available. Besides this peculiar arrangement of the appendix the cæcum, into which it opens, forms a kind of swelling or diverticulum at the head of the colon, and fæcal matter has here also a tendency to become lodged. These conditions explain the frequency of inflammatory lesions of the appendix, which may be primary, but is more frequently due to the presence of small irritant bodies, such as pips of raisins or other fruits, cherry stones, shot, small biliary calculi, &c. When these foreign bodies remain a long time in the vermiform appendix they generally become covered by a layer of tribasic phosphate and form calculi. Calculi of this nature have been found which contained

centrally a mass of ascarides' eggs (Lücke). When inflamed, either spontaneously or in consequence of the presence of foreign bodies (which is most frequent), the mucous membrane of the vermiform appendix secretes a puriform or mucous fluid ; it becomes thicker, more or less vascular, and the closed follicles it contains often ulcerate. The entire appendix becomes distended, and its walls much thicker than normally; the infiltration and thickening of its mucous layer render contraction of its muscular coat impossible, and consequently entirely prevent its emptying itself or changing its place. It therefore remains for some time in the situation it occupied at the commencement of the lesion. As the inflammation very frequently extends to the peritoneal investment of the appendix, it becomes covered by a delicate layer of fibrin, which is traversed by blood vessels and newly-formed connective tissue; hence it results that the immobilised appendix contracts adhesions with the parts in relation with it. This peritonitis is from the commencement adhesive and limited, and is generally not serious. It undergoes resolution, leaving the vermiform appendix bound to the neighbouring organs by complete or filamentous adhesions. It most frequently unites it to the cæcum throughout its whole extent, when it undergoes atrophy. At other times it becomes united to the uterus, to the bladder, or to the abdominal wall, but then only by its free extremity ; whence it comes that when the band of adhesion has become solid by fibrous organisation of the new tissue a kind of ridge may be formed, beneath which the small intestine may become strangulated. An ulcer, which may begin in the mucous membrane of the appendix, may invade its whole wall and extend into the inflammatory tissue which unites it to the neighbouring organs. It is thus that abscesses of the iliac fossa are formed, which show a tendency to extend outwards beneath Poupart's ligament. Fistulæ are formed in this way which may communicate or not with the cæcum. Klebs has seen a fistula formed by mucous membrane and a fibro-serous envelope, which communicated between the vermiform appendix and the rectum.

Proctitis.—It is rare for the colon to be inflamed throughout its whole extent. The descending portion and the rectum only are the most frequently attacked. Proctitis, or inflammation of the rectum, is primary in sporadic or epidemic dysentery. It often follows hæmorrhoids (hæmorrhoidal catarrh), the arrest of foreign bodies in the depressions of the mucous membrane, syphilitic affections of the anus and rectum, rhagades, mucous plaques, and

tertiary syphilitic ulcers. In cancer of the uterus, even when the walls of the rectum are unaffected by the neoplasm, more or less acute catarrhal inflammation of the mucous membrane is generally present.

Dysentery.—Dysentery is an ulcerative inflammation of the large intestine. It is generally located in the rectum and the transverse and descending colon; when very acute the whole colon may be affected. The various forms of this malady, namely, the sporadic dysentery of temperate climates, the sporadic and epidemic dysentery of Algeria and tropical countries, and the chronic diarrhœa of the latter, resemble one another entirely in respect to their anatomical lesions, and only differ by their cause and their acuteness. The lesions are, however, not identical at the commencement of the disease (acute dysentery) and during its period of chronicity; hence acute dysentery will first be described and then chronic dysentery.

1. **Acute dysentery.**—In the mild or catarrhal form of acute dysentery the surface of the mucous membrane is very red, congested, and covered by small ecchymoses. These lesions, which extend throughout the rectum and the descending colon, are particularly marked at the folds of the mucous membrane, which is itself thickened and granulating. The follicles project. The mucous membrane is covered by a slight mucous exudation, resembling white of egg, which may be purulent in places, and is generally streaked with blood and is uniformly stained pink or red by blood. This exudation constitutes the dysenteric stools, which are mucous, glairy, and resemble frog's spawn, are slight in amount, often streaked with blood, and frequently passed, defecation being accompanied with tenesmus and a burning sensation at the anus, which with the other characteristics suffice for the recognition of dysentery. A few days after the commencement of the attack small punched-out ulcers are seen on the mucous membrane; they are covered by a transparent or cloudy mucus, but when this is removed the ulcer looks deeper than it really is, owing to thickening of the mucous membrane. These lesions are very limited in simple dysentery, and rapidly undergo cure in our climate.

Microscopical examination of the thickened mucous membrane reveals the following conditions :—(1) In the glandular layer the blood vessels are turgid, and the connective tissue surrounding them is infiltrated with lymph cells; the interglandular septa

are also increased in thickness and in length in consequence of the inflammatory infiltration. The glands of Lieberkühn are elongated, and as they are compressed alternate dilatations and contractions are observed. The epithelial cells of the glands are preserved, and are generally hypertrophied and cup-shaped. As to the epithelial cells of the surface, it is useless, as we have already said, to look for them 24 hours after death; but it may be considered certain that they are partially detached during life, for they are found in the dysenteric stools at the commencement of the disease. (2) In the submucous connective tissue the lymph cells are extremely numerous around the blood vessels. The connective tissue of the mucous membrane is equally infiltrated with lymph cells beneath the glands of Lieberkühn and around the closed follicles. The latter, filled with lymph cells, are tumefied and project on the mucous surface; they finally soften centrally, empty themselves of their contents, suppurate, and give origin to a follicular ulcer. When the inflammation is very acute and the exudation, containing lymph cells and fibrin, infiltrates the connective tissue to the point of compressing the blood vessels, an actual mortification of a limited portion of the mucous membrane occurs. Suppurative eliminative inflammation soon then occurs around the mortified parts. More or less extensive flakes of the glandular layer are detached by the subjacent suppuration, and are expelled in fragments, the true nature of which is more or less recognisable by the naked eye or under the microscope. They are found in diarrhœic evacuations. When mortification extends to the glandular layer a deep and more extensive ulcer is formed with a flat base and irregular edges; it is generally seated at the top of a fold. When a lymph follicle and the surrounding tissue are affected, a small round and deeper ulcer is produced. These ulcers, once formed, continue to extend by breaking down of the parts infiltrated with lymph cells; they secrete pus all the time that the dysentery continues in the acute stage. This may undergo cure by repair of the losses of substance, by the formation of granulation tissue and consequent cicatrisation, or it may pass into the chronic condition.

The lesions of acute dysentery, which occurs sporadically or in epidemics in hot countries, are similar, but the type of the disease is much more acute, and the lesions extend to the larger part, or the whole of the large intestine; and more or less extensive catarrh of the mucous membrane of the small intestine may be coexistent. At the post-mortem examination of patients who

have died during the acute stage of epidemic dysentery the ulcers, described above, are found, but are larger, deeper, and extend over the whole surface of the colon from the cæcum to the anus. The surface of the ulcers is sometimes covered by débris infiltrated with pus, not yet detached from the superficial glandular or mucous layers; sometimes it is red or pink, granulating and brown, or grey from decomposition of the blood. The edges of the ulcers are sloping, bordered by a swollen, congested, ecchymotic, and softened mucous membrane. These ulcers may become so extensive that only a few islets of intact mucous membrane may remain, the rest being completely destroyed. Thus in an examination which M. Kelsch made in the laboratory of one of us, in the course of a very interesting research on dysentery ('Arch. de Phys.' 1873), three-quarters of the whole length of the colon was ulcerated; on the smooth, pink, uniform, ulcerated surface, patches about the size of a five-franc piece were seen from place to place, formed of a very friable fungoid tissue of a much darker red colour than the ulcerated parts. These represented all that remained of the glandular layer. In the preserved portions of the mucous membrane the glands of Lieberkühn are found with their cylindrical cells; but they are deformed, irregular, here compressed, there distended; the blood vessels surrounding the glands are distended and gorged with blood. Around them the connective tissue contains lymph cells and numerous filaments of fibrin. The subglandular connective tissue, which forms the base of the ulcer, is itself infiltrated with inflammatory exudation composed of round cells and fibrin; the blood vessels are much dilated and full of blood, and their walls have returned to the embryonic condition. The lymph vessels contain large swollen endothelial cells. The submucous cellular tissue is the seat of true phlegmon; it has doubled or trebled in thickness, and its most superficial part, beneath the glands, is changed in places into a pool of pus, which isolates the glandular layer and renders its destruction inevitable. Thus it is not rare to see patients evacuate with the stools large pieces of mucous membrane, either in flakes or cylinders, which may be many inches in length. It seems needless to add that dysentery attended with lesions so profound and extensive terminates fatally; death occurs either from exhaustion or, which is rarer, from perforation of the intestine and subacute peritonitis, or as the result of hepatic abscess.

Examination of the stools in acute dysentery gives the following results: At the commencement they are composed of a small

quantity of glairy, vitreous matter in pellets, which resemble mucous sputa. They have been compared to frog's spawn. They are often blood-stained, either uniformly or in streaks. They contain lymph cells, red blood corpuscles, cylindrical cells, mucous cells, and numerous microbes. Such are the stools of the first stage. In the period of commencing ulceration the stools are composed of a serous, blood-stained fluid, in which are suspended white membranous fragments, varying in size; sometimes actual cylinders, which are composed of the superficial part of the necrosed mucous membrane infiltrated with pus and detached. On examining these flakes under the microscope portions of the glands of Lieberkühn may be recognised, or even a group of glands united together. This reddish diarrhœic liquid mixed with white débris has been likened to shreds of sodden meat. When the necrosed parts have been eliminated, the stools are composed only of an ichorous fluid, purulent or serous in character and grey or blood-stained in colour. This fluid, which is secreted by the ulcers, contains a large number of lymph cells and red blood corpuscles.

The other abdominal organs are often secondarily affected in dysentery; the bladder is generally congested and the seat of acute catarrh; the kidneys are often affected with catarrhal or parenchymatous nephritis; the lumbar lymph glands are congested and hypertrophied, and the spleen is large and soft; and, finally, congestion and abscess of the liver is often found. The small intestine sometimes shows traces of a more or less acute catarrh; at other times it has collapsed, as well as the stomach, and a remarkable atrophy of its coats may be observed.

2. **Chronic dysentery.**—Chronic dysentery is either consequent on acute dysentery or occurs spontaneously. A fatal termination occurs only at the end of several months, often of many years. At the autopsy the mucous membrane is seen to be swollen and congested at places. The raised edges of depressions which simulate ulcers at first sight are very red. In these depressions, which look like losses of substance, the glands of Lieberkühn are found to be preserved; at other points true ulcers are present. These implicate either only the superficial portion of the glandular layer or penetrate to the submucous cellular layer. The surface of these ulcers is brown or slate-coloured; in it more or less regular orifices may be seen with the naked eye, which open into follicular depressions situated deeply in the submucous connective tissue. From these small cavities, which exist in considerable numbers and which measure from ½ to 3 mm. in diameter, a

concrete mucus resembling frog's spawn may be squeezed out by pressure. This gelatiniform mucus, examined in the fresh state under the microscope, is seen to contain cylindrical cells and chalice cells, which are arranged around the edge of the small drop parallel to one another. The centre of the same fluid mass contains long fusiform ovoid or round cells, which are nothing else than cylindrical cells or white blood corpuscles in a mucoid condition. This mucus, treated by nitric acid or acetic acid, gives an opaque precipitate.

Hardened sections of the mucous membrane, comprising the edge of an ulcer, the ulcer itself, and the cavities full of mucus, show the following conditions:—(1) In the swollen and con-

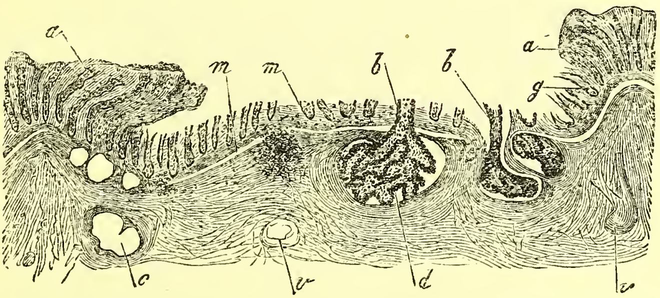


FIG. 111.—VERTICAL SECTION OF THE INTESTINE PASSING THROUGH A DYSENTERIC ULCER.

a, a, projecting portions of the mucous membranes with tubular glands; *m, m*, tubular glands reduced to their culs-de-sac situated in the ulcerated part of the mucous membrane; *b, b*, losses of substance filled up with mucus and cylindrical cells opening by a narrow orifice on to the surface of the mucous membrane; *d*, wall and contents of the hiatus; *v*, vessels. Magnified 20 diameters.

gested mucous membrane which separates the ulcers (*a*, fig. 111) the glands of Lieberkühn are seen to be very long and large; they are separated by connective tissue through which run blood vessels distended with blood. Beneath the glandular layer the superficial muscular layer of the mucous membrane is normal, and the connective tissue is simply hyperæmic; at the same time the cells are larger than in the physiological condition. The follicles are seen in section to be sometimes elliptical in shape, the long diameter being parallel to the mucous membrane, at others circular. (2) At the depressed and ulcerated parts the mucous membrane only shows vestiges of the tubular glands. They are reduced to the lower third of their length and are entirely wanting in places. The partly destroyed saccules contain cylindrical cells which are mostly cup-shaped. The glands

are separated from one another by connective tissue infiltrated with lymph cells, which is continuous with the equally altered connective tissue of the subglandular layer. In the ulcerated parts it looks as if the superficial layer of the mucous membrane had been shaved off, the glands and the fibrous tissue separating them having been destroyed at the same time. In the parts where the tubular glands are destroyed the connective tissue which is in direct relation with the contents of the intestine does not seem to be lined by epithelial cells. (3) The follicular de-

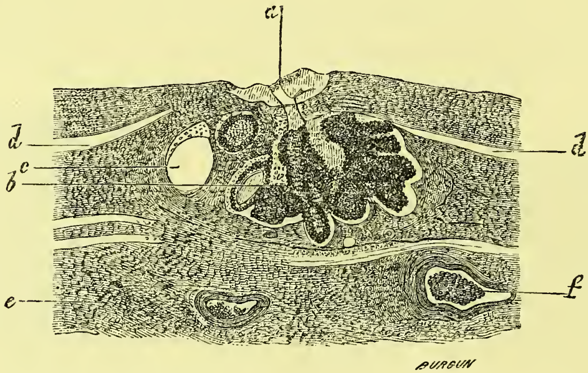


FIG. 112.—VERTICAL SECTION OF AN ULCERATED INTESTINE, AT A SPOT WHERE THE TUBULAR GLANDS HAVE BEEN COMPLETELY DESTROYED BY ULCERATION.

b, loss of substance filled with mucus and cylindrical cells; *a*, orifice, or neck of the loss of substance opening on to the surface of the intestine; *c*, compartment empty; *d*, superficial muscular layer of the mucous membrane interrupted at the orifice, *a*; *e*, connective tissue; *f*, vessel. Magnified 40 diameters.

pressions or losses of substance, visible to the naked eye and filled with mucus, always open on to the ulcerated surface. In shape they are generally elliptical or round, their long diameter being always parallel to the surface of the mucous membrane. They open by a perfectly round orifice, varying in diameter (*a*, fig. 112). The muscular layer of the mucous membrane is interrupted at these orifices, but it may be traced above the small cavity between it and the surface of the mucous membrane. Sometimes there is one single cavity (*d*, fig. 111). Sometimes the cavity is formed of many compartments completely or incompletely separated from one another by fibrous septa (*c*, fig. 112). Both the single cavity and the multiple compartments are filled with mucus and bordered by a layer of cylindrical cells. These mucous contents are easily detached from the fibrous wall; they insinuate themselves into the depressions or irregularities of the hiatus. The mucous

mass contracts into the form of a cork in the neck of the follicular cavity; it then spreads out on the surface of the mucous membrane (*a*, fig. 112). All these structures taken together may at

FIG. 113.—VERTICAL SECTION OF THE INTESTINE THROUGH AN ULCER.

g, inflammatory embryonic tissue, in which tubular glands, *e*, may be seen irregularly dilated; *a*, large round cavity or loss of substance with irregular edges, the wall of which, *b*, is lined at certain parts with cylindrical epithelium, *f*. Magnified 100 diameters.

first sight simulate a racemose gland. The wall of these cavities is formed of connective-tissue cells, between the fasciculi of which turgid connective-tissue cells and leucocytes are found. On their internal wall a complete lining of cylindrical cells is seen implanted vertically, as on a mucous membrane. Sometimes the epithelial cells are absent over a part of the cavity. Thus in fig. 113 the epithelial lining of the large cavity, *a*, is only seen at the portion marked *f*. The cylindrical cells which line these cavities are nearly all cup-shaped (see *a*, fig. 114). In the cavities where they are only present over a part of the surface they are only observed on the upper part. At the spots where the cylindrical cells are absent the wall is generally very rich in lymph cells, and acute destructive inflammation is present, which prevents the formation of the epithelial lining (*vide* c, fig. 114). The connective tissue of these cavities is infiltrated with lymph cells for a more or less extensive zone. At this spot the tubular glands have disappeared or are reduced to their culs-de-sac, or they have become hypertrophied and dilated, so that their lower extremity,

filled with cylindrical cells, is divided into two or three buds (*vide* fig. 113, *e*).

We believe that these cavities, full of mucus, generally occupy

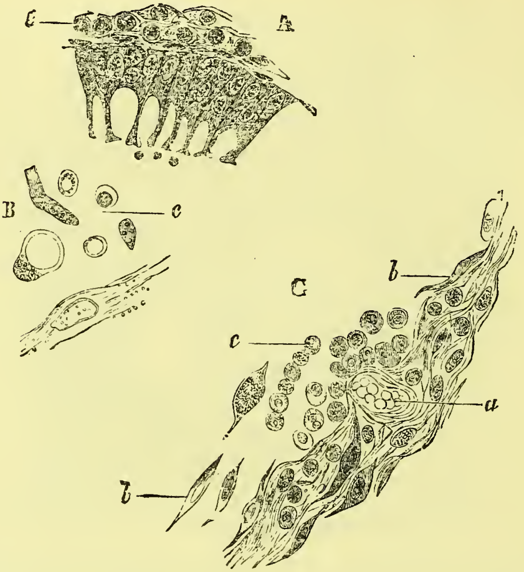


Fig. 114.—A Few Spots in the preceding Figure Magnified by a Power of 400 Diameters.

A, cup-shaped cylindrical cells lining the wall of the large cavity represented in fig. 113 ; *e*, lymph cells infiltrating the tissue bordering the cavity.
C, part of the wall of the same cavity in which no cylindrical cells are seen ; *c*, lymph cells free in the mucus ; *d*, flat cells equally free ; *a*, blood vessel filled with red blood corpuscles.
B, free cells in the mucus which have become vesicular.

the place of destroyed closed follicles, and we base our opinion on the following facts : They have the same seat, the same relations with the glandular layer and superficial muscular layer, and the same form as the closed follicles ; further, we have seen closed follicles undergoing softening and destruction. The latter are larger at first than normally, and after the lymph corpuscles have been brushed out of them the retiform tissue will be seen to be wanting in places, and many large compartments are found, limited by trabeculæ. When these softened parts of the inflamed follicles have been eliminated from the surface of the mucous membrane we have the septate cavities already described, cavities which are soon filled with intestinal mucus and lined by cylindrical epithelium similar to that on the surface of the intestine or in the glands. Once formed, these cavities, occupying

the place of the closed follicles, increase by destruction of the remaining septa, and may dilate till they attain a diameter of from 4 to 5 mm. (These changes, the naked-eye appearances of which have been well described by many writers, and particularly by Charcot, were first, we believe, determined by one of us, and were shortly afterwards studied by Kelsch.) The submucous connective tissue is also thickened at the same time, becomes fibrous, and its lymph vessels are found to be filled with swollen endothelium. The inflammation often extends to the connective tissue which separates the muscular layers of the intestine, and even to the subserous tissue. This fibrous thickening of all the layers of the large intestine, marked more particularly in the cellular layer, causes apparent hypertrophy of the muscular coats, and has the result of changing the intestine, particularly at its lower part, into a tube with almost rigid walls. This is, moreover, what is often seen in chronic dysentery with rather extensive ulcerations which have undergone cure and have been replaced by a dense and solid cicatricial tissue. On these cicatrices, where the mucous membrane is never completely reconstituted, polypoid excrescences are often found, composed of connective tissue and fibro-mucous polypi.

The evacuations vary much during the course of chronic dysentery; generally they are serous, abundant, of a yellow, greenish, or brown colour; but if an acute attack supervenes they become mucous and contain blood or a little pus. Diarrhœa is not always constant during the whole course of the malady; it may be suspended temporarily, to return later.

Cholera.—Cholera is an infectious disease, the most important manifestation of which consists in serous diarrhœa corresponding to a lesion of the small intestine. At the autopsy of subjects who have succumbed during the algide period the mucous membrane of the small intestine is found congested throughout its whole extent, more particularly marked in the ileum. It is of a pink, lilac, or red colour; the staining is more marked at the summit of the *valvuli conniventes*, owing to repletion of the capillaries and small veins. The mucous membrane is thickened, turgid, and œdematous. The intestine is distended by a large quantity of white, cloudy, inodorous fluid, similar to that voided during life, and in which small, opaque, flocculent masses (rice-water evacuations) are suspended. The superficial epithelium of the villi and of the mucous membrane in contact with this fluid

naturally falls after death, but examination of the choleraic stools during life leads one to think that the cylindrical epithelium of the intestine does not become freely detached during the choleraic attack. The cloudy state of this liquid is due not only to the presence of lymph cells, but chiefly to the existence of a considerable number of micro-organisms, such as are met with in fluids undergoing putrefactive changes. Pacini in the first instance, and Davaine later, pointed out the presence of a large number of microphytes, micrococci, bacteridia, and vibrios in choleraic stools. Chemical analyses of the evacuations show that they are poor in organic substances. They contain 3 or 4 per 1,000 of albumen (Becquerel). Urea, or its product of decomposition, carbonate of ammonium, is found in them and renders the fluid alkaline. The proportion of chlorides and other salts is the same as in normal intestinal fluid. Mechanical analysis of cholera stools closely resembles that of the liquid obtained by Moreau in the experiments described above. Very soon after the invasion of cholera, at the commencement of the algide period, the closed follicles of the mucous membrane of the small intestine, particularly those at the lower part of the ileum, are tumefied, and are seen as small round and prominent granules of a pinkish grey or grey colour.

The changes in the mucous membrane and the mucous corium are found, when examined microscopically, to be much more profound and acute than would be supposed by gross appearances. They were described by Kelsch and Renaut in the epidemic of 1873. The connective tissue of the mucous membrane is infiltrated with very numerous lymph cells. This infiltration is present in the fibrous septa which are found between and beneath the tubular glands. It is not limited to the small intestine, but occurs throughout the whole length of the intestinal tube, from the pylorus to the anus. It is also present to a variable extent in the intestinal villi. The glands of Lieberkühn are generally deprived of epithelium, except in the lower part of their saccules (a lesion which is partly post-mortem), and they are often distended with mucus. The blood vessels of the superficial part of the mucous membrane are dilated and filled with blood, and it is the same with the vessels of the mucous corium. The lymph vessels are filled either by round cells or by some of their own endothelial cells, which are swollen and detached. The closed follicles show the same changes described above when considering psorenteria. Centrally they show some tendency to softening, and their cellular elements are infiltrated

with fat granules. The muscular coat is normal, but the subserous connective tissue is hyperæmic and infiltrated with lymph cells. Irritation of the peritoneum may even be present, which is shown by the delicate false fibrinous membranes which cover its surface. At a more advanced stage, when the subject has succumbed during the period of reaction in an acute attack of cholera, follicular ulcers are sometimes found, seated either in the isolated follicles or in Peyer's patches. The mucous membrane is less congested; persistent hyperæmia is, however, present, and sometimes superficial ulcerations are found in the folds of the small intestine, or irregularly on the top of the folds of the large intestine. These ulcerations may extend into the deeper tissues, so as to nearly cause perforation (Hamernyck). In other cases the intestine is stained, as if washed and atrophied. The physical characters of the contents of the intestine differ entirely from those observed in the first period. There is no longer the rice-water fluid free of gas, which is present at the commencement. The intestinal fluid is stained by bile and of a brown colour, and it frequently contains blood. The colon contains hard fæcal matter, or on the contrary fluid fæces. The changes in the blood in cholera are very marked during the first or algide period. The blood is wanting in serum and is frothy, so that it circulates with difficulty, and stasis may even occur. From the considerable diminution of blood serum it results that the number of red blood corpuscles for the same amount of blood is two or three times larger than normally. The number of leucocytes is increased in the same degree. The red corpuscles are viscous and diffluent; a large number of them seem smaller than normally, their diameter being, for example, 3 to 4 μ instead of 7 μ. There seem to be no special proto-organisms in the blood of cholera subjects. Renal lesions are observed in cholera with albuminuria, the nature and cause of which will be studied in the chapter on renal diseases. In the period of reaction in cholera, the serum of the blood gradually regains its normal amount, and the number of red corpuscles diminish proportionately. It is, however, probable that the blood corpuscles are destroyed in great numbers in this period of the disease, for the urine contains a large amount of colouring matter. In the same period the blood contains a large amount of urea, and congestive and inflammatory lesions are produced in various organs, such as pulmonary congestion, bronchitis, laryngitis, and pleurisy, which latter is sometimes purulent. Congestion, ecchymoses, and œdema of the pia mater are

also observed. Suppuration of the parotid gland, cystitis, and pyelonephritis are seen more rarely.

To recapitulate: cholera seems to be caused, as Pacini long ago sought to establish, by a micrococcus which penetrates into the epithelial cells of the intestine, multiplies there in great numbers, and causes desquamation of the epithelial lining. The intestinal villi, which are more or less denuded, allow the transudation of a large amount of blood serum through the capillary network. This view of Pacini explains most of the phenomena observed in cholera, particularly the sero-albuminous intestinal secretion, and the concentration of the blood. This theory equally supports the contagiousness of cholera, which seems to us to be indisputable.

Uræmic ulcers.—In the colon, and more rarely in the lower part of the small intestine, ulcers are sometimes found, which were first described by Treitz, and which he referred to uræmia. They are preceded by catarrh, and by fluid stools which are alkaline and contain a rather large amount of carbonate of ammonia. At the beginning of the ulceration the stools contain a little blood and débris from the mucous membrane. The ulcers are, in fact, consecutive to limited gangrene of the mucous membrane and elimination of the eschars. It is a kind of gangrenous dysentery in which the intestinal mucous membrane is not either notably thickened or congested, but shows eschars followed by ulceration. These ulcers arise in the closed follicles and the surrounding tissue, and they extend so that they may attain many centimetres in size, their long axis being generally longitudinal; they vary in number; they may cicatrise and leave a smooth grey surface.

Typhoid fever.—The intestinal lesions of typhoid fever are seated at the lower part of the small intestine. They are rarely seen in the colon. Four successive states may be distinguished in the lesions, which correspond to the four periods of the disease; they are: (1) catarrhal state of the intestinal mucous membrane; (2) swelling and ulceration of Peyer's patches; (3) the formation of eliminative eschars; (4) cicatrisation.

1. In the first period, which generally lasts four or five days, the mucous membrane is congested, and secretes a variable quantity of liquid, which causes a more or less abundant diarrhœa. The isolated closed follicles and Peyer's patches are tumefied from the commencement of the disease, particularly at the lower

part of the ileum. The isolated follicles form small beaded projections, and are pink and semitransparent, as in cholera (psorenteria), and the Peyer's patches show a marked relief.

2. The hypertrophy of the isolated follicles and Peyer's patches increases. At the autopsy of patients who die at the fifth or sixth day of typhoid fever, in the serious form of the disease when delirium is present from the beginning, it is true that we have seen the isolated follicles in the form of hard conical and projecting papules, measuring 3 or 4 mm. from their apex to their base, and the Peyer's patches thickened in the same way. These are the lesions which Louis has described under the name of *hard patches.* They seem to correspond to the most serious of the clinical forms of the disease. During the second week the Peyer's patches which are nearest the cæcum, that is to say, those which are the first and the most seriously attacked, already begin to ulcerate partially at one or two spots in the same patch, while in the upper part of the ileum their surface is regular and not ulcerated. On dividing an isolated follicle with a scalpel, it is seen to be composed of white, grey, or slightly pink tissue, and to be of a soft consistency. Thus the name of medullary tissue has been given it by comparing it to brain substance. On scraping this tissue a small amount of cloudy fluid is obtained. On closely observing the divided surface no marked limit is seen between the follicle and the mucous membrane, so that it might be thought at first sight that pathological infiltration occurs simultaneously in the closed follicle and in the connective tissue which surrounds it. Peyer's patches show similar lesions, only that the infiltration is general; all the follicles, or a large number of them, as well as the neighbouring connective tissue, are affected at the same time. The number of altered patches varies greatly: sometimes there are only two or three, in which case they are those nearest the ileo-cæcal valve; sometimes 20 or 30, or even 50, may be counted. When the lesion is more marked the entire patch is hypertrophied and greatly thickened; it may, for example, project 2 or 3 mm., sometimes even more, and its surface shows on section that each one of the follicles composing it is formed of grey tissue and full of juice. Patches tumefied and hypertrophied to this degree are, again, the *hard patches* of Louis.

When the lesion is less marked, the isolated follicles and the patches are not so prominent; the patches are only tumefied partially, and a small number of the follicles are affected (*soft patches* of Louis). The hard patches and the closely acuminated

follicles, which resemble in their naked-eye appearances the papules of variola, lend themselves best to microscopic study. It is useless to look for superficial epithelium in situ in the villi or on the surface of the patches, for the autopsy not being made till twenty-four hours after death, the cylindrical cells are all detached, and form a mucous, opaque, puriform fluid, which covers the surface of the mucous membrane. On scraping the divided surface with a scalpel small fragments of the grey substance of the patch can be removed; and on examining these in picro-carminate a considerable number of lymph cells will be seen, some of which contain one nucleus and others several smaller nuclei. In this fluid other larger cells may also be found, which are round or polygonal, with blunt angles, flat or tumefied, with a granular protoplasm, and contain one or more oval nuclei. These large cells are the tumefied and inflamed endothelial cells of the retiform tissue of the closed follicles and of the lymph sinuses. They resemble the cells observed in leukæmic products of the spleen and in lymphadenomata. They have been called typical cells, and have been regarded as peculiar to typhoid fever, but they have in reality nothing characteristic.

In order to study in situ the changes which the various parts of the intestine undergo, flakes of the diseased part should be hardened in absolute alcohol or Müller's fluid, gum and alcohol, after extending them on flat pieces of cork. Sections passing through Peyer's patches which have not ulcerated show the following details: *a.* The villi are enlarged, at the same time that they seem to be shortened; they show a tendency to approach one another, and their bases to become blended. This change in shape is due to the fact that the tissue of the villi is infiltrated with lymph cells. This effacement of the villi becomes so marked that under the microscope the surface of the patch seems scarcely villous, and to the naked eye it appears quite smooth. *b.* The glands of Lieberkühn increase in length and width in passing from the normal part of the mucous membrane to the diseased patch. In fig. 115 is seen a section of the normal mucous membrane, at a certain distance from a diseased patch, and in fig. 116 is shown the mucous membrane of an hypertrophied Peyer's patch. These two figures, drawn under the same power, show the differences existing in the two parts of the mucous membrane. In the first the villi are very distinct and the glands small: in the second the villi are swollen and seem to be melting together at their bases; only their extremities are distinct.

The glands of Lieberkühn are two or three times longer than normally, and their transverse diameter is increased in the same proportion. The cylindrical epithelial cells of the hypertrophied glands are longer than in normal glands, and their lumen contains

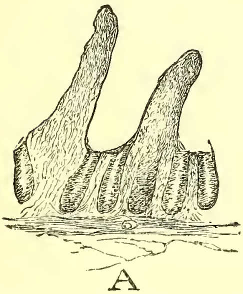


Fig. 115. — Portion of the Intestinal Mucous Membrane near to a Peyer's Patch, from a case of Typhoid Fever. Magnified 40 diameters.

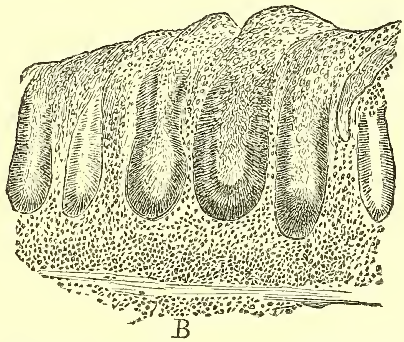


Fig. 116.—Portion of the Intestinal Mucous Membrane situated at the Edge of an Hypertrophied Peyer's Patch at a point where no Closed Follicles were present, from a case of Typhoid Fever.

The cylindrical epithelial cells are preserved and are very large in the tubular glands. These glands are on an anterior plane, and the villi on a much deeper plane; hence they appear in the figure as if continuous with the glands, which they are not. Magnified 40 diameters.

round cells or deformed cylindrical cells. The connective tissue situated around and beneath the glands is infiltrated with small round elements, and is directly continuous with the villi which are diseased in the same way. The lesions of the villi and glands are the same whether in Peyer's patches or at the level of the isolated follicles. *c.* The deep layer of the mucous membrane, which forms the largest part of the hard patch, appears at first sight to be homogeneous. It is crossed by blood vessels which are considerably distended and filled with red and white corpuscles. The latter are greatly in excess of the normal number. On brushing sections, cut after hardening in picric acid, the contents of the vessels and the majority of the lymph cells just mentioned can be removed. These cells were contained in meshes of the reticulated tissue of the lymph follicles and of the connective tissue. This tissue, thus cleared of the free elements it contained, shows large circular spaces, which, by the arrangement of the enclosed retiform tissue and the condensed layers which circumscribe them, are easily recognised as lymph follicles.

Such is the structure of hypertrophied patches in typhoid fever; it will be seen that it consists essentially in infiltration of the adenoid tissue and of the connective tissue of the mucous membrane with lymph cells.[1] The superficial tubular glands are hypertrophied at the same time, and the lesions they show seem to be simply caused by changes in the connective tissue which surrounds them. In this second period of the disease ulceration always commences in the patches and isolated closed follicles situated nearest the ileo-cæcal valve. The process of ulceration can be easily observed with the naked eye in hard patches; it is the result of the mortification of a more or less extensive portion of either a patch or an isolated follicle. The most prominent part of the follicle or patch which is necrosed becomes of a yellowish colour, which staining is due to the intestinal juice. The circulation is arrested; it is at first circumscribed by a line, then by a groove, and finally the tissue is eliminated in small fragments. Side by side with one of these small eschars which is still in situ others may be quite eliminated; they are replaced by an ulcer, the base of which gradually becomes clean. Fresh points of necrosis occur successively in hard patches, and are followed by ulceration. At the commencement of the process the isolated follicles present a small eschar at their most prominent point, which on being eliminated is followed by an ulcer situated at first in the centre of the follicle, but which enlarges by progressively invading the whole structure. Such ulcers often show a great tendency to extend downwards, and having only a small diameter, they may even perforate the muscular coat and the peritoneum. At the level of a hard patch the visceral peritoneum is red, and all the vessels, particularly the small veins, are dilated and filled with blood. When the patch ulcerates the serous membrane covering it is thickened, and shows opaque grey or whitish spots, slightly prominent, and which might be mistaken by their naked-eye appearances for tubercles.

After commencing in the mucous membrane the inflammation is propagated downwards, and the connective tissue which separates the two layers of the muscular coat and the serous membrane

[1] Small localised epidemics of typhoid fever, the cases reported by Murchison, and particularly by Budd, demonstrate the infectious character of the disease. Everything supports the view that the contagious agent is transmitted by the drinking water, and that it consists of micro-organisms. According to Klebs the microbe of typhoid fever is a bacillus which makes Peyer's patches, the closed follicles, the mesenteric glands, and the spleen its habitat.

are often infiltrated with lymph cells. In delicate sections of the intestine cut perpendicularly to the peritoneum, a large number of lymph cells are in fact seen in the peritoneal connective tissue

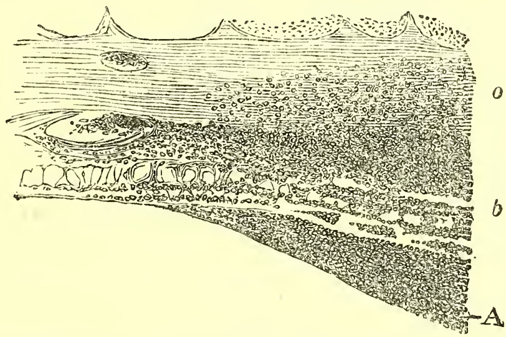


FIG. 117.—SECTION OF THE SUPERFICIAL MUSCULAR LAYER AND OF THE PERITONEUM OF THE SMALL INTESTINE AT THE LEVEL OF AN HYPERTROPHIED PEYER'S PATCH IN TYPHOID FEVER.

A, new formation on the surface of the peritoneum; *b*, subperitoneal connective tissue infiltrated with lymph cells; *o*, muscular tissue similarly altered. Magnified 80 diameters.

and in the layer of longitudinal muscular fibres (fig. 117). At the level of the white spots which are visible to the naked eye on the surface of the peritoneum a number of embryonic cells are collected together in an amorphous ground substance. This superficial inflammation of the peritoneum, which resembles tubercular infiltration at first sight, is distinguished from it by the absence of distinct granulation and caseous degeneration. This new formation is located on the surface of the peritoneum, and is directly covered by the endothelium of this membrane. Infiltration of all the intestinal coats by cells, and consequent softening of the connective-tissue bundles, are the conditions which favour ulceration, its extension on the side of the peritoneum, and perforation of the intestine. The soft patches and hypertrophied follicles generally ulcerate in the same manner as the hard patches, only that the eschars are less exactly defined, and molecular destruction and elimination of the superficial parts seem to be what takes place more than the formation of actual eschars. In the case where the different follicles composing a Peyer's patch are separately affected by ulceration, the patch shows an irregular surface covered with depressions (*reticulated patch* of Louis). In sections of these reticulated patches, made after repair has commenced, it is seen that the layer of villi and tubular glands is partly preserved, thereby showing that the

ulcerative and destructive process has not been complete. Rather frequently one or more follicles are seen transformed into small abscesses, from which a drop of serous pus can be squeezed on opening them. If from a hardened specimen a section be made

Fig. 118.—Section of a Lymph Follicle the Centre of which is converted into a Small Abscess, from a case of Typhoid Fever. At the upper part of the Figure Capillary Vessels and Cells are seen free in the Cavity of the Abscess.

passing through one of these small follicular abscesses, the elements of the pus are removed by the mode of preparation, and an empty space is seen in the middle of the follicle ; this space is bordered by the débris of capillary vessels and trabeculæ of retiform tissue (*vide* fig. 118). The wall of these small follicular abscesses is formed of connective tissue, the fibres of which are granular and allow of rows of lymph cells being seen between them.

3. **During the third period**, which corresponds to the third week of typhoid fever, the ulcers of the plaques become clean by the elimination of all tissue much infiltrated with lymph cells. The least diseased tissue, which forms the walls and bases of the ulcers, is highly congested and forms small fleshy granulations. The walls of the embryonic vessels are friable, and hæmorrhage easily occurs. It is, in fact, in this stage that serious intestinal hæmorrhage occurs, and at the autopsy of persons who have died in consequence rents in the vessels may be discovered through which the blood has been poured into the intestine. At the level of these rents small ecchymotic points are observed and an opening is distinctly visible. Rupture of the vessels and hæmorrhage during the second week are observed much more rarely, though they are possible during the separation and elimination of the necrosed parts.

4. **At the end of the third period the process of repair commences** and is continued during the **fourth week.** Pink-coloured fleshy granulations spring up from the base of the ulcer and gradually attain the surface. The tissue composing these fleshy granulations condenses, the edges of the ulcer approach, and a cicatrice is formed. But cicatricial repair goes on slowly, so that in autopsies made six weeks, two months, or even more after the commencement of the disease small ulcers may still be found. The cicatricial tissue is almost always pigmented black, and this pigmentation persists, so that years afterwards Peyer's patches are recognisable which had been at a former period affected by typhoid lesions. In a case of typhoid fever which had been cured three months previously, but in which convalescence had been marked by the symptoms of relapse, we observed at the autopsy very acute catarrh of the whole of the digestive tube, and one of the patches was still partially ulcerated ; the others had cicatrised with irregular and pigmented surface. Histological examination in this case showed us that the mucous membrane at the level of the formerly ulcerated patches had been replaced by connective tissue with longitudinal fibres separated by a large number of interposed round cells, and that there did not remain a vestige of the closed follicles, the glands, or the villi. The blood vessels had remained large, dilated, and with embryonic walls, and around them as well as on the surface of the mucous membrane black pigment was present. Typhoid fever ulcerations do not cause subsequent narrowing of the intestine.

The *mesenteric lymph glands* are constantly altered in typhoid fever. They are at first turgid, hypertrophied, and congested, then grey or pinkish grey; they then become infiltrated by a considerable quantity of lymph cells. On cutting them across and scraping the divided surface a fluid is obtained which contains, besides lymph cells, large tumefied endothelial cells containing one or more nuclei. It is not very rare to find, in subjects who have succumbed to typhoid fever, glands showing spots or foci of pus which may extend to the whole cortex. On examining sections of such glands under the microscope their capsule is seen to be infiltrated with lymph cells continuous with the peripheral connective tissue, which is equally inflamed, and in which fat cells are replaced by embryonic cells. Beneath the capsule the afferent lymph vessels are often filled and distended by a fibrinous coagulum, the meshes of which contain a considerable number of lymph cells. The follicles are rather large, and within them the

arterioles are surrounded by zones of proliferation; finally, the perifollicular lymph sinuses and the cavernous tissue contain a large number of swollen and proliferating endothelial cells. The capillaries and small veins are filled with red blood corpuscles. We have here, in fact, inflammatory lesions similar to those of the closed follicles of the intestine. The mesenteric glands sometimes contain, as the result of typhoid fever, caseous masses which are caused by the transformation of inflammatory products.

The *spleen*, which is constantly affected in typhoid fever, is hypertrophied, gorged with lymph cells, pink or red, but more frequently pale and soft. The Malpighian corpuscles, when visible, are generally larger than usual. The *large intestine* is rarely affected; when implicated its follicles are tumefied, and the thickened mucous membrane forms patches or small round tumours. These patches, similar to those in the small intestine, are seated either in the cæcum, the colon, or the rectum; they also become ulcerated. The *liver* and *kidneys* are almost invariably affected by parenchymatous inflammation, such as is always observed in infectious febrile diseases. We shall return to this subject when considering diseases of the liver and kidneys. With regard to the *stomach* the reader is referred to p. 246. The *muscles* often, though not always, undergo fatty or vitreous degeneration. The adductor muscles of the thigh, and those of the abdominal walls, the recti, for example, are most frequently affected. The cardiac muscle does not escape granulo-fatty degeneration more than the others, and this morbid change is sometimes coincident, during the later period of the disease, with marked weakness of the systole and intermittence of the heart beat. We must finally note congestion and inflammation of the *lungs*, which is constant in this disease. Besides congestion and bronchitis, catarrhal pneumonia, atalectasia, pulmonary infarctuses, and even acute lobar or fibrinous pneumonia may sometimes be present. At the autopsy of patients who have succumbed during the course of the disease, or while convalescing, pulmonary tubercles have been found, thus showing, contrary to the opinion of certain authors, that there is no antagonism between typhoid fever and tuberculosis. The *brain*, the *spinal cord*, and their membranes are sometimes congested. Such are the lesions of typhoid fever, which is a disease *totius substantiæ* as every other infectious febrile disease, but of which the principal lesions are located in the intestine.

Lesions of the intestine in herniæ.—We do not propose to

describe here the different kinds of hernia, the study of which belongs to surgery rather than to pathological histology, but we shall give a short *résumé* of what is known of lesions of the intestine in inflamed strangulated hernia.

In incarcerated or inflamed hernia the sac does not always contain fluid; at other times it contains a serous transparent fluid, of a pink, blood-stained, or blackish colour, and in which fibrinous false membranes are present. The intestinal serous membrane is sometimes quite normal, sometimes very red, as in acute peritonitis. It may be opaque and show false membranes, or even vascular fleshy granulations. The condition of the mucous membrane has not been studied in a complete manner; it is seen to be red. Small abscesses have been observed between the coats of the intestine. If the hernia is not reduced the inflammation ends in fibrous adhesions between the sac and the intestine, and by thickening of the sac and the intestinal membranes.

In strangulated hernia the colour of the serous surface of the intestine is of a deeper red than in inflamed hernia. It is ecchymotic, brown in colour, approaching violet or black. The hernial loop is tense, and filled with fluid and gas during the first period of strangulation. The tense peritoneum shows slits or erosions at the strangulated parts, and the subserous cellular tissue is the seat of ecchymoses. Later on the peritoneum is covered by a fibrinous exudation, and the peritoneal sac contains a more or less blood-stained fluid. The strangulated loop contains a rather abundant mucous fluid, which may be blood-stained, or quite hæmorrhagic in character; gas is present in small quantities, but fæcal matters are very seldom found. The mucous membrane is deeply congested from the first, and soon shows the signs of acute inflammation of all its parts. The villi are tumefied, softened, friable, and longer than in the normal condition; a false membrane may be formed on the surface which may hide them completely. The isolated and agminated closed follicles are hypertrophied, infiltrated with juice, and ulcerated at their centre. These lesions are particularly marked at the seat of strangulation, and chiefly at the spot where the strangulated loop is continuous with the upper part of the intestine, which is itself dilated and distended with fæcal matter, particularly with gas. The inflammation of the mucous and serous membranes extends, and often becomes the starting point of general peritonitis. The portion of the intestine which is situated below the strangulated intestine is decreased in

size and contracted, and it is also the seat of inflammatory lesions of the mucous and serous membranes, less marked, however, than those at the upper extremity. When constriction at the ring persists, the hindrance to or arrest of the circulation produced causes ulceration of the intestinal membranes at this spot. This ulceration takes place without gangrene; all the coats of the intestine seem to be cut through mechanically (Gosselin). The ulceration commences in the superficial layer of the mucous membrane, and progressively invades the submucous tissue, the muscular coat, and finally the peritoneum (Nicaise), thus causing perforation, which is sometimes extremely limited in area, sometimes, on the contrary, implicating most of the circumference of the intestine. Another and much rarer termination to intestinal strangulation is gangrene which is seen at different points of the strangulated loop in the form of superficial patches, or which affects simultaneously all the coats of the intestine.

Internal strangulation.—The lesions observed in internal strangulation, &c., are the same generally as those described in hernia; inflammation and gangrene are equally present. In *volvulus* the upper part of the small intestine, or the large intestine, slips into the part immediately following, so that there is invagination of the upper part in the lower. The small intestine may also penetrate thus into the colon. A tumour is consequently produced in which the first intestinal envelope is in relation with the second by its mucous membrane, while the middle one is in relation with the internal by the serous membrane. Mucous polypi are sometimes, particularly in children, the cause of invagination by reason of their weight. Invagination may undergo cure by expulsion of portions of the gangrenous intestine, and by subsequent cicatrisation of the two ends of the intestine; but it may also be followed by perforation, peritonitis, abnormal adhesions, and almost always, when cured, by cicatricial narrowing of the intestine.

Hypertrophy of the muscular coats of the intestine.—In certain forms of obstinate constipation with retention of fæcal matter both the small and large intestine are found after death, particularly in old subjects, to be considerably distended. When this condition has lasted a long time with various changes, the dilated intestine, instead of being thinned, shows considerable hypertrophy of its walls. This hypertrophy is chiefly of the muscular walls. It is

similar to hypertrophy of the muscular coats of the stomach in consequence of stenosis of the pylorus.

Rectal fistulæ.—Fistulæ caused by periproctitis, that is to say, by inflammation of the connective tissue surrounding the rectum, may be divided according to their seat into two varieties: 1st. Those found in the sacro-sciatic notch and in the inferior pelvi-rectal space, and which follow suppurative inflammation of the adipose tissue, which is so abundant in this region. They are almost complete; that is to say, they open on one side into the rectum below the levator ani muscle, and on the other after a more or less long and complicated course on to the skin. 2nd. Superior pelvi-rectal fistulæ which are almost always blind externally (Pozzi); that is to say, they have an external opening, but none on the side of the rectum. They consist generally of an upper sac which secretes pus, and which is situated above the levator ani muscle. They always make their way high up the rectum, often 10 or 15 c.m. above the anus, and they are separated from the rectum by indurated connective tissue. Both these forms of fistulæ present the common character of being hollowed in the midst of connective tissue, modified by chronic inflammation, in which fat cells have disappeared, and the fasciculæ are separated by tumefied connective-tissue cells and lymph cells. The fistulous tracts themselves, when quite recent, form pouches, or are at least irregular and bordered by suppurating fleshy granulations; when older, the fistulous tracts are lined with mucous membrane covered by pavement epithelium. They show a few distinct papillæ furnished with blood vessels and covered with stratified epithelium. These fistulæ are frequently met with in tubercular subjects.

Tubercle of the intestine.—The tubercular lesions of the intestinal mucous membrane are generally seated, as in typhoid fever, in the lower part of the small intestine, but they are not confined to this area, and they generally extend to the ileum, the jejunum, and to the colon even as far as the rectum. These lesions are characterised by tubercular granulations, caseous and ulcerative inflammation of the isolated and agminated lymph follicles, and by infiltration of the surrounding connective tissue. From their starting point in the mucous membrane they extend to the deeper layers and invade the subserous connective tissue, the lymphatics of the intestine, and even those of the mesentery. We shall describe together tubercular granulations and tubercular inflam-

mation of the closed follicles, for the two lesions are almost always contemporaneous, tubercular inflammation of the follicles sometimes preceding the formation of tubercle. Tubercular granulations of the intestinal mucous membrane commence as small round, semitransparent granules, projecting on the surface, and which may be located either in the connective tissue around the saccules of the glands of Lieberkühn or in the connective tissue of the villi. In some experiments in which Chauveau produced tuberculosis in bovine beasts by the ingestion of tubercular lungs, he was able to observe the commencement of tubercular granulations on the surface of the intestine; and some of these he saw develop in intestinal villi which were tumefied and infiltrated with small round cells. We have also observed very small granulations on the surface of the mucous membrane and in the villi in man; in sections the connective tissue of the villi was seen to be infiltrated with round cells. These villi, swollen throughout their whole extent and particularly at their base, were more or less blended together, and were only distinct at their free extremity. They formed, together with the equally infiltrated stroma of the mucous membrane, a nodule which appeared both to the naked eye and under the microscope to have the structure of a tubercular nodule. The tubular glands, compressed and choked by this new formation, showed a series of changes bearing chiefly on the cylindrical cells. They were small, irregular, and mixed with lymph cells, and all had undergone caseous change. When tubercular granulations commence in the deep layers of the mucous membrane they are covered by a layer of glands and villi. The connective tissue of these villi and that which separates the glands of Lieberkühn are equally infiltrated with lymph cells, and at a later stage they participate in the formation of granulations. In tuberculosis, the special inflammation of the closed follicles does not, at the commencement, differ from simple psorenteria; but the centres of inflammation soon become larger, opaque, and whitish or yellowish in colour; they are larger and harder than tubercular granulations. Often, on pricking them with the point of a needle, a little whitish or yellow cloudy fluid is emitted, which contains lymph cells in a more or less granular condition, and rather large round cells containing two or three nuclei and fat granules. On examining these small tumours in hardened sections it is seen that at their periphery they are more sharply separated from the surrounding tissue than tubercles. Finally, if the centre of the follicle has been occupied by a small

abscess, the pus which has become coagulated by the action of the hardening fluid will be detached *en masse*, leaving an empty space in the middle of the follicle. These small follicular abscesses open into the intestinal cavity, empty out their contents, and are replaced by ulcers which continue to increase in size. These diseased isolated or agminated follicles and the neighbouring tissues thickened by inflammation form a projecting plaque, which soon undergoes ulceration at one or more points where the follicles are most altered. It is in this way that large ulcerating patches are formed in tuberculosis. On examining the edges of these ulcers under the microscope in sections cut perpendicularly to the surface of the mucous membrane, almost identical lesions are found in the villi and glands as on the surface of tumefied patches in typhoid fever. The villi are in fact partly effaced by swelling; they are infiltrated with embryonic elements and changed into large fleshy granulations; the glands of Lieberkühn are changed in form, compressed at certain points, while they are dilated at others, particularly at the level of the saccules. They are filled with cylindrical cells, which are generally larger than normally. At the edges of ulcers which are suppurating freely, the villi are seen to be changed into very vascular fleshy granulations,

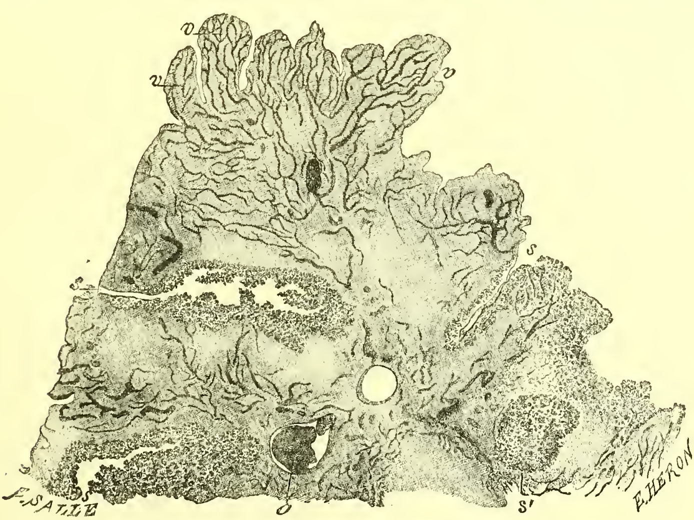


Fig. 119.—Section of a Tubercular Ulcer in the Intestine.
v, *v*, villi preserved and changed into fleshy granulations; *s*, *s*, deep grooves and pits lined with cylindrical epithelium; *s'*, transverse section of one of these depressions. Magnified 20 diameters.

separated from each other by deep pits which are lined with well-preserved cylindrical epithelium (fig. 119). When tubercular

granulations are confluent at any one spot in the intestine, around them, as around inflamed follicles, irritative lesions of the mucous membrane and submucous tissue are caused, and an ulcer soon makes its appearance at the points primarily affected, where the circulation has been arrested. The ulcer is most frequently produced by molecular gangrene. Whatever may be the starting point of the ulcer, whether it may be primarily due to granulations or to tubercular inflammation of the follicles, its appearance, its ulterior development, and its consequences are the same.

Tubercular ulcers of the small intestine are generally seated at the lower part of the ileum in Peyer's patches; they are circular or elliptical in form, their long axis being longitudinal when seated in Peyer's patches; those, on the other hand, located elsewhere, in the jejunum, colon, or even in the ileum, have generally their long axis transverse to the direction of the intestine. Rindfleisch thinks that this latter form, which is the most frequent, is due to the fact that tubercular granulations invade by preference the walls of the blood vessels and lymphatics, the direction of which is perpendicular to that of the intestine. The edges of the ulcer are sinuous, serpiginous, and prominent, and contain either tubercular granulations or diseased and caseous follicles. Their base is equally sprinkled over with grey or whitish nodules, which for the most part correspond to tubercles undergoing elimination.

The part played by the lymphatics in the development of intestinal tuberculosis can be easily made out. On examining the peritoneal surface, which corresponds to an ulcer of the mucous membrane, a certain number of fine tubercular granulations will always be observed; they are semitransparent or slightly opaque at their centre. The lymph vessels which emerge at this spot, and are directed towards the mesenteric glands, look like knotted cords of a white or yellowish white colour. From point to point are noticed prominences formed by tubercular granulations developed in their walls, and, on dividing these across, a whitish, caseous substance can often be squeezed out. This matter is composed of swollen granular endothelial cells and lymph corpuscles. These various elements are often in a state of granulo-fatty degeneration; hence the opacity of the mass. Sections of these vessels made from hardened specimens show the walls to be infiltrated with round cells and to contain here and there tubercular granulations, at which spots they are considerably hypertrophied. These granulations are surrounded with injected

blood vessels; their centre is granulo-fatty, and their elements have the same arrangement as in every other tubercle. The

FIG. 120.—TRANSVERSE SECTION OF A TUBERCULAR LYMPH VESSEL FROM THE PERITONEAL SURFACE OF THE INTESTINE.

a, cavity of the lymphatic, which is contracted and filled with lymph cells; *e*, sub-peritoneal connective tissue, through which passes the lymphatic, the walls of which are much thickened by development of tubercular tissue; *l*, tubercular tissue, the external zone of which is formed of round cells larger than those of the internal zone, which are caseous; *v*, capillary vessels; *n*, layer of longitudinal smooth muscle fibres; *m*, layer of transverse muscle fibres of the intestine.

lumen of the vessel is generally contracted and irregular; it has the form of a long or ovoid slit, and it is filled with granular lymph cells (figs. 120 and 121). The various layers of the

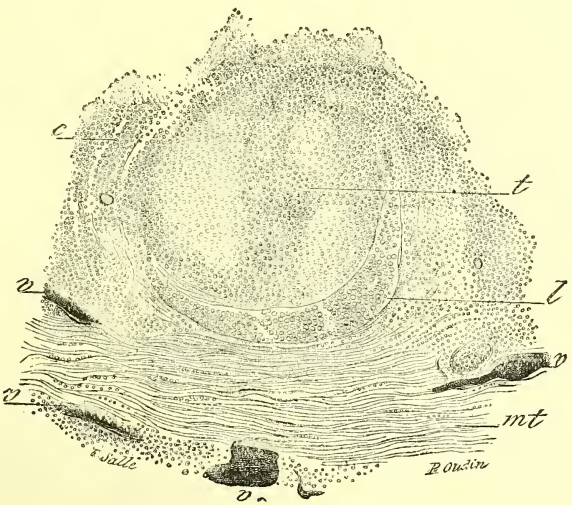


FIG. 121.—SECTION OF A TUBERCULAR GRANULATION DEVELOPED IN THE WALL OF A LYMPH VESSEL.

t, granulation; *l*, cavity of the lymph vessel full of cells; *v*, *v*, blood vessels; *mt*, section of longitudinal muscle fibres. Magnified 80 diameters.

connective tissue of the intestine may be the seat of granulations. They are found in the mucous corium, between the two muscular layers, or in the substance of one of these, the muscular fasciculi being dissociated by the new formation (fig. 122). As the ulceration increases the deep layers of the intestine undergo more and more marked inflammatory changes, and new groups of tubercles are formed. This complex tissue — embryonic tissue sprinkled over with tubercular granulations—is itself very liable to suppuration and molecular gangrene; thus the ulcer, which is always increasing, may end in perforating the intestine; but this lesion is rare, for tubercular subjects affected with intestinal ulceration generally succumb before the lesion has reached its final stage. Sometimes inflammatory and tubercular infiltration of the mucous membrane may produce such considerable thickening as to cause narrowing of the intestine. We have seen a case of this kind in which the narrowing was seated at the end of the ileum. Klebs relates a similar case of tubercular narrowing of the ileo-cæcal valve. It is unnecessary to add that in tuberculosis of the intestine the mucous membrane is the seat of more or less acute catarrh with hyper-secretion of fluid and consequent diarrhœa.

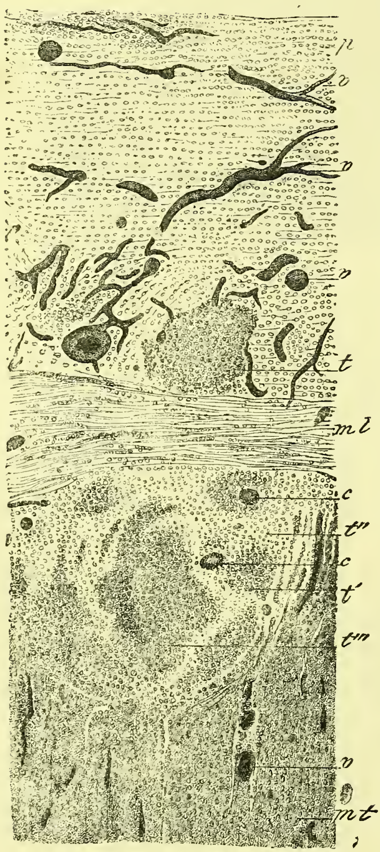


FIG. 122.—SECTION OF THE INTESTINE AT THE LEVEL OF A TUBERCULAR ULCERATION.

p, peritoneum infiltrated with lymph cells, supplied with blood vessels, *v*, and showing a tubercular granulation, *t*; *ml*, layer of longitudinal muscle fibres; *mt*, layer of circular muscle fibres. Between these two layers a tubercle, *t' t''*, is seen, and giant cells, *c*, *c*. Magnified 80 diameters.

The tubercular ulcerations just described are consecutive to ulcerating tubercle of the lung, or they may be due to the fact that patients swallow their sputa. Tubercle primarily developed in the peritoneum rarely gives origin to intestinal

tuberculosis and ulcerations. On the other hand, tubercular ulcerations of the small intestine always cause an eruption of miliary granulations on the corresponding surface of the visceral layer. This eruption enables one to distinguish the ulcers of tuberculosis from those of typhoid fever. The tubercular granulations of the peritoneum, which correspond to ulceration of the mucous membrane, are distinguished as small semi-transparent granules in a red patch; the granulations at the periphery of the patch are more numerous and larger than those at the centre. At this spot peritonitis is sometimes present with false membranes and adhesion of an intestinal loop with neighbouring loops. General peritonitis is rarely produced.

Syphilitic tumours and ulcers.—Ulceration of the intestine following syphilitic gummata is not much more frequent than that of the stomach. There are, however, well-authenticated cases reported by Cullerier, Fœrster, Meschede, Wagner, Eberth, and Klebs. In the case of Eberth, which was that of a new-born infant, there was pemphigus and gummata of the thymus and lungs. The loops of the small intestine were united by fibrous bands in which caseous nodules were found. The case related by Klebs was that of a man thirty-six years of age, who died from syphilitic ulceration of the skin and larynx, gummata of the liver and lungs, and ulcers of the stomach and of the small and large intestine. Near the ileo-cæcal valve there was an ulcer a foot long. In the small intestine and colon the smaller isolated ulcers measured from 1 to 3 c.m. across. The lowest of them was situated a little above the anus. The edges of these ulcers were thick; they often showed nodules which were caseous centrally. Their base yielded a little pus, and was formed of a dense yellowish grey tissue, which was fibrous in consistency; it resembled cicatricial tissue, and projected on the serous side. Under the microscope this tissue seemed to be formed of a connective-tissue substance, infiltrated with small cellular elements. This change commences in the small intestine in the lymph follicles of Peyer's patches. It is probable that it also commences in the large intestine in the isolated follicles, but it soon invades the surrounding connective tissue. The lymphatics which ramify on the surface of the intestinal serous membrane at the level of these ulcerations are altered and knotty.

The *rectum* is the seat of extensive ulcerations accompanied

with narrowing, which is sometimes very marked. This is known by the name of *syphilitic stricture of the rectum.* According to Gosselin, Bærensprung, Leudet, Perret, and Lancereaux, this narrowing is not due to syphilis; but this view is denied by other pathologists, among whom is Fournier. The ulcerations which precede the stricture commence a few centimetres above the anus and are sharply defined; they exude an abundant secretion of pus. These ulcers differ from those of chronic dysentery in that they are only found at the lower part of the rectum, and that they cause narrowing, whereas dilatation of the rectum is, on the contrary, more frequently observed in chronic dysentery. They are more common in women than in men, and follow either chancres of the anus, mucous plaques, or condylomata, which morbid growths are frequent in this region.

Fibroma.—Fibroma is rarely developed on the surface of the intestinal mucous membrane; small fibrous growths in the form of polypi are found in the chronic forms of dysentery around ulcers of the rectum, and at the anus, where they take the form of papillomata. A formation of fibrous tissue mixed with adenoma is seen in certain mucous polypi which show a development of papillæ on their surface.

Myoma.—In the muscular coats of the intestine small sessile or pediculated tumours are sometimes found: they are composed of fibrous tissue and smooth muscle fibres. They do not differ from myomata of the stomach. Small lipomata may project under the intestinal mucous membrane, but they are rare; on the other hand lipomatous polypi frequently project into the peritoneal cavity from the ascending, transverse, and descending colon; they may be looked upon as much enlarged *appendices epiploicæ* of the large intestine.

Vascular tumours.—Dilatation of the veins, or phlebectasia, is rather frequent in the large intestine, particularly in the hæmorrhoidal plexus, where it constitutes the tumours known as *hæmorrhoids.* Varices projecting under the mucous membrane may always be found in the small intestine. These dilatations are caused by anything which determines blood stasis in the afferent branches of the portal vein: cirrhosis, cardiac disease, abdominal tumours, increase of size of the uterus in pregnancy, the strains of parturition, constipation and the efforts of defæcation, &c.;

these are all causes which impede the course of the blood in the hæmorrhoidal veins.

Hæmorrhoids consist at the beginning in simple dilatation of the veins at the edge of the anus, which then project slightly. Simultaneously the veins under the rectal mucous membrane, above and below the sphincter, become dilated. Later they become folded on themselves, and form vascular tumours which swell during defæcation. Rupture of these dilated veins and hæmorrhage follow, and catarrhal irritation of the mucous membrane of the lower part of the rectum is generally produced. The veins contained in the hæmorrhoids show the lesions of varices ; the intervening connective tissue thickens, becomes indurated, and unites the veins together. Phleboliths are sometimes found in their walls.

Adenoma.—These tumours, characterised by simple hypertrophy of the glandular layer, are rarely met with in the small intestine, but are very frequent in the rectum, where they form *mucous polypi.* These tumours, which are often observed in children, are soft and vascular, and on section show a decidedly

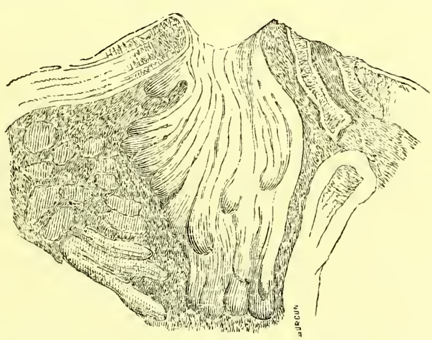


Fig. 123.—Transverse Section of a Small Polypoid Adenoma of the Small Intestine. Magnified 20 diameters.

glandular appearance. In microscopic sections the glandular tubes will be seen pressed one against another. The epithelial cells on the surface of the tumour are similar to those in the intestine. The glandular tubes contain cylindrical epithelium, between the cells of which a varying number of caliciform cells are found. Sometimes the tubes show cystic dilatations lined with caliciform epithelium containing mucus. Generally the connective-tissue stroma of these tumours is slight in quantity,

and the tubes seem to be in contact. These polypi may be the cause of prolapsus of the rectum. If they project beyond the anus the epithelial layer in contact with the air changes its character; the superficial cells, which were cylindrical like those

FIG. 124.—TRANSVERSE SECTION OF A MUCOUS POLYPUS OF THE RECTUM.

b, free surface of the polypus ; *m*, cylindrical-celled epithelium lining the glandular tubes and cavities, *c* ; *d*, fibrous trabeculæ ; *v*, vessels. Magnified 20 diameters.

of the intestine, become polygonal, and, by being superimposed, form a pavement epithelium. This peculiarity is observed in other polypi which become external.

Lymphadenomata.—Lymphadenomata of the intestine have already been described in the first volume of this Manual (vol. i. p. 245). They are chiefly found in the lower part of the small intestine, near the ileo-cæcal valve, but they may also be found throughout the whole length of the gastro-intestinal tract. When attacked by this new growth the ileo-cæcal valve may form an actual swelling. These tumours vary greatly in volume ; they are seen in the form of nummular patches, more or less extensive, more or less projecting and confluent, depressed, or ulcerated at their centre. They invade the mucous membrane throughout its whole thickness, and are found equally well in the parts free of isolated or agminated follicles as in those where they are present. They may infiltrate the muscular coat of the

intestine, and, blending with the mesenteric glands, form an enormous mass. They are generally coincident with similar growths developed in the glands, spleen, peritoneum, lungs, bones, and skin. In the latter case fungoid mycosis or cutaneous lymphadenia is produced, the nature of which affection was first determined by one of us (*vide* Gillot's thesis). In a case reported by Béhier the lesions of the intestine seem to have been single. Lymphadenic tumours of the intestine have the naked-eye appearances of encephaloid carcinoma; as in carcinoma, they yield a milky juice, and their extent and thickness, the progressive invasion of the neighbouring tissues and degeneration of the lymph glands, make the resemblance still closer. Though they sometimes commence in the closed follicles and Peyer's patches, lymphadenic infiltrations and ulcerations differ entirely in their naked-eye appearances from those present in typhoid fever. They are much larger, and they extend indifferently to all parts of the mucous membrane. Microscopic examination leaves no doubt as to the nature of the growth; the numerous lymphatic elements which the tumours contain, the large-meshed reticulated stroma, with its distinct trabeculæ easily seen on brushing out the cells, constitute unmistakable characters.

Carcinoma.—Primary carcinoma of the intestine is incomparably rarer than that of the stomach, but it takes quite the same form in both portions of the digestive tract. It is often secondary to carcinoma developed in the neighbouring organs, the peritoneum, bladder, uterus, &c. The parts of the intestine most frequently affected by carcinoma are the rectum, the transverse colon, and the angle of the colon which is in relation with the liver; the cæcum, the duodenum, the ileum, and the jejunum are much more rarely affected. Scirrhus and encephaloid are met with in the rectum, the first more frequently than the second; they develop either at the sphincter or 8 or 10 centimetres above; they originate in the submucous connective tissue. The infiltration extends in a circular manner around the intestine, and causes more or less marked stricture. The granulating appearance of the mucous membrane, its ulceration, the extension to the deep layers, hypertrophy of the muscular coats of the rectum, and propagation of the infection to the lymphs glands, are phenomena similar to those seen in carcinoma of the stomach. Mucous carcinoma of the rectum is thought to be very common; it is certain that colloid tumours of the rectum and of the rest of

the intestine are rather frequent, but it must not be concluded that every colloid tumour with large alveoli filled with a gelatinous substance is a carcinoma. We have already pointed out how cylindrical-celled epithelioma may undergo colloid change, and hence the most recently formed parts of the tumour should be examined before deciding as to its nature. Carcinoma primarily developed in the mucous membrane generally forms a single tumour which grows in every direction, and causes one or more strictures, which may, however, disappear, like stricture of the pylorus, by the progress of ulceration. The ulcerating tumour finally causes the destruction of the intestinal wall at a certain point, and perforation results. This accident is rarer in the intestine than in the stomach. Carcinoma is rarely seen in the intestine as a multiple tumour; nevertheless one of us saw a case in which a large number of colloid carcinomatous tumours were present throughout the whole length of the small intestine. The tumours were ovoid, not ulcerated, projected into the intestine, and were developed from the mucous membrane and submucous connective tissue. Primary carcinoma of the intestine produces secondary growths in the peritoneum, liver, lungs, kidneys, &c. The nearest lymph glands are always affected.

Epithelioma.—Cylindrical-celled epithelioma is one of the

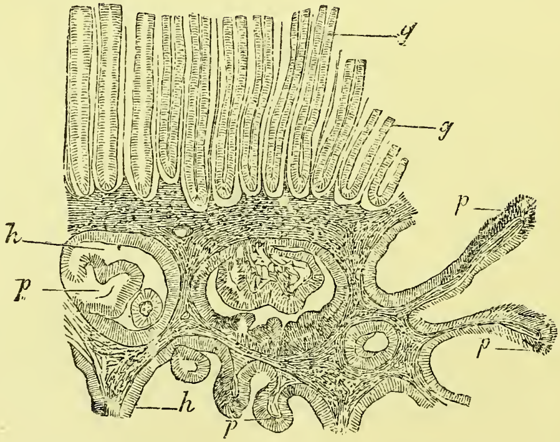


Fig. 125.—Section of a Cylindrical-Celled Epithelioma of the Large Intestine.

g, g, layer of hypertrophied glands of Lieberkühn. These glands are destroyed to the right, where ulceration has taken place. *h, h*, cavities lined with cylindrical epithelium. From the walls of these, papillæ, *p, p*, are thrown out, which are also lined with cylindrical cells. At the ulcerated part to the right the papillæ, *p, p*, are very long and lined with cylindrical cells.

commonest tumours of the small intestine, the colon, and the rectum. It has exactly the same gross appearances, the same form, and the same microscopical details as epithelioma of the stomach. As in that organ, it commences by hypertrophy of the tubular glands; the muscular layer of the mucous membrane is then perforated, and the saccules penetrate into the submucous tissue, where they form cavities lined with cylindrical cells; from these cavities vascular buds equally covered with cylindrical cells are often given off. These epithelial cylinders and expansions of the glandular saccules then penetrate into the muscular layers of the intestine. Part or the whole of the morbid mass often undergoes colloid degeneration. Pavement, horny, or mucous epithelioma is often found near the anus; these tumours do not differ in structure from similar epitheliomata observed in other parts of the body.

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